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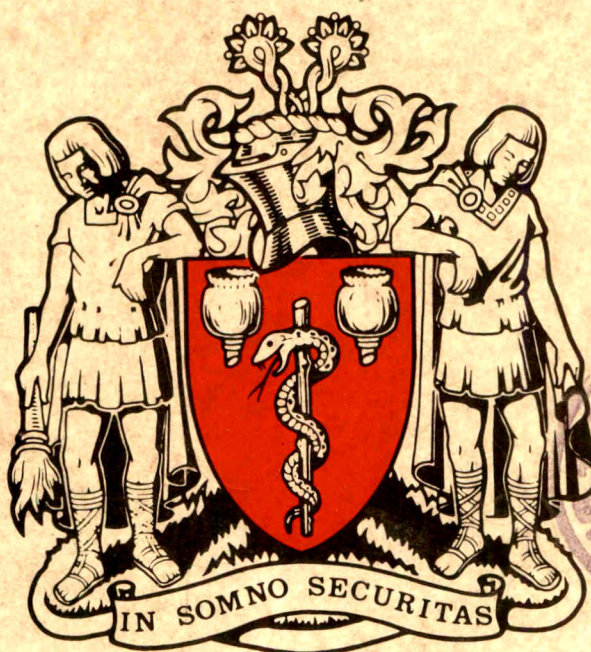
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Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 45 Number 9 September 1990

CUR-HO2483-15-P024532



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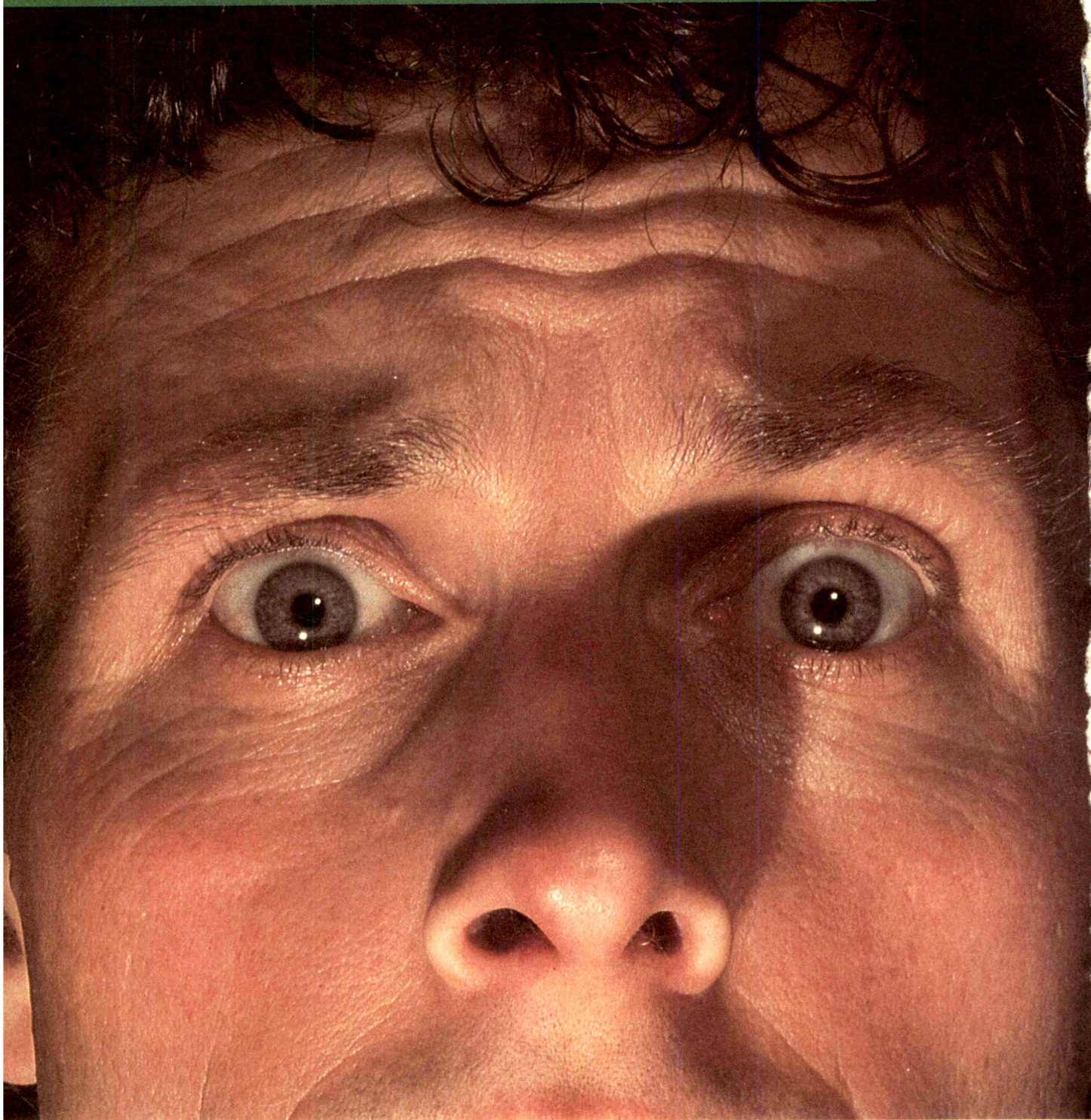


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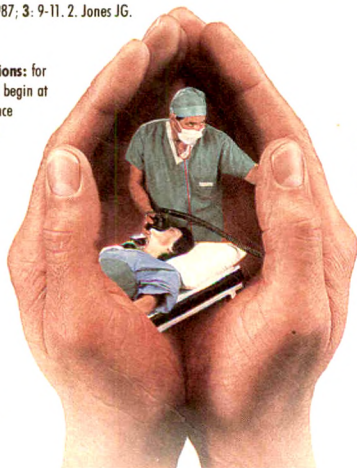
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ANAESTHESIA: ISSN 0003-2409, Volume 45 1990, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

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Second class postage rate paid at Jamaica, NY 11431, USA.

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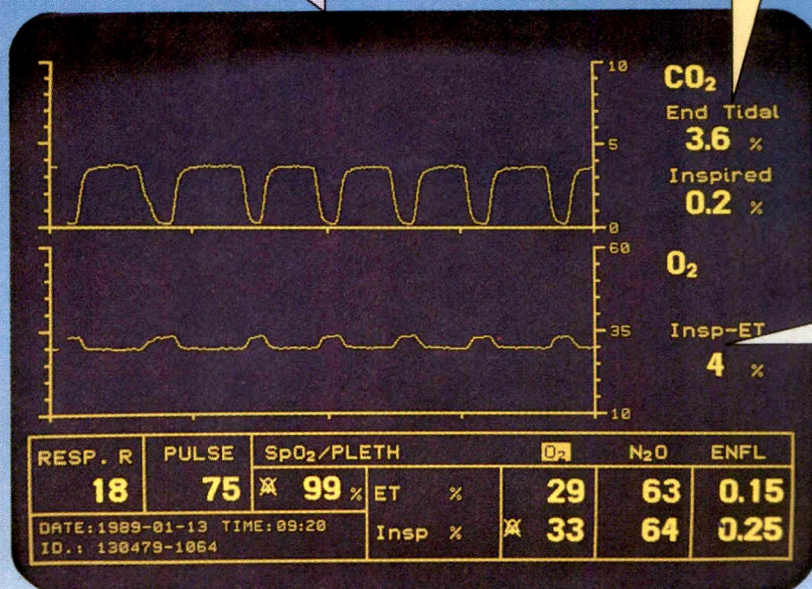
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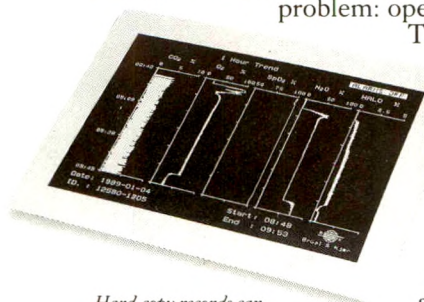
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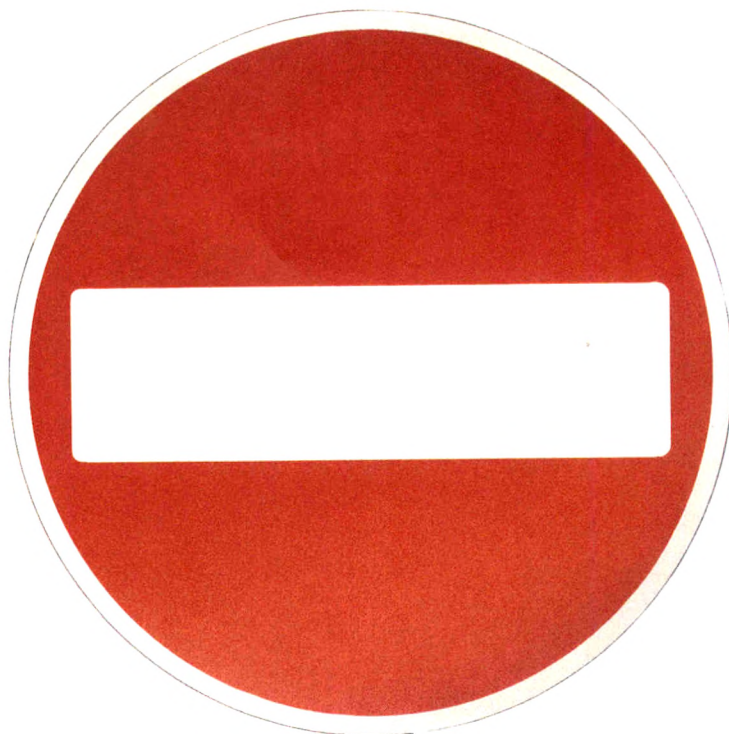
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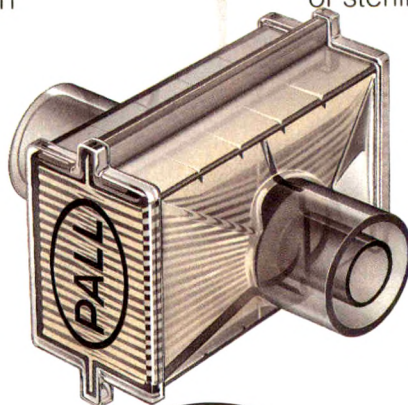
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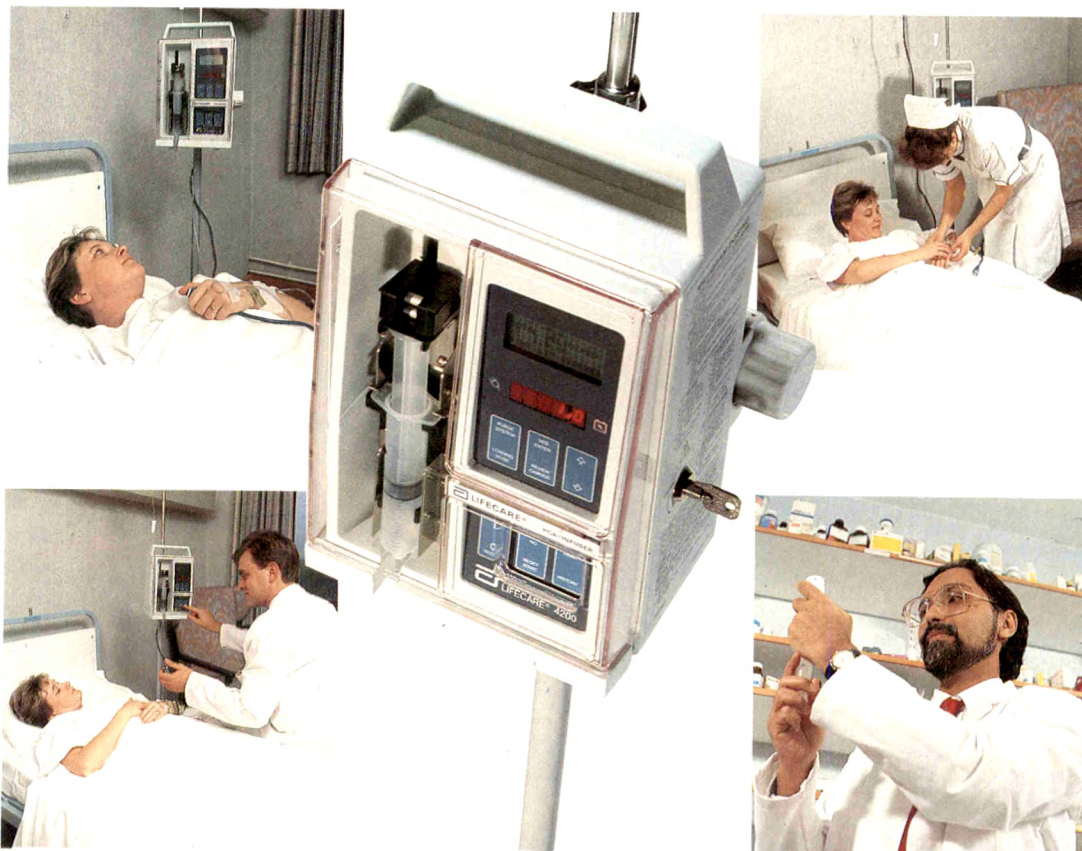
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Editorial

Anaesthesia for cataract surgery—time for change?

The recent paper on local anaesthesia for eye surgery—the peri-ocular technique—highlighted the involvement of the anaesthetist, the simplicity and success of the method and the paucity of complications.¹ Almost all cataract operations are performed under local anaesthesia in the USA, and the total hospital stay rarely exceeds 2 hours. That choice may be influenced by the fact that medical insurance carriers will not cover inpatient cataract surgery under general anaesthesia except in specified circumstances. However, we should consider whether local anaesthesia provides adequate anaesthesia and good operating conditions and whether we should continue to use general anaesthesia for the majority of cataract extractions in the United Kingdom. There are few studies that compare the efficacy and anaesthetic and surgical complications with the different anaesthetic methods²⁻⁴ and prospective controlled studies are awaited eagerly. Moreover, there seems to be a reluctance by some British surgeons to operate on awake patients who might be more likely to move during a sophisticated microsurgical operation. There also appears to be relative satisfaction with general anaesthesia for an operation that, although mostly performed on elderly, frequently unfit people, causes relatively few problems during or after operation. However, there is now evidence that cataract surgery under general anaesthesia evokes an endocrine and metabolic response that can be prevented completely by local anaesthesia⁵.

Local anaesthesia for cataract surgery is administered mostly by eye surgeons: the method of combined retrobulbar and facial blocks is often used. These cases are usually put on separate operating lists (or at the end of the list) ostensibly so that the anaesthetist can be spared for other duties. There are obvious disadvantages to the operator/anaesthetist concept. Occasionally, serious complications have occurred, including retrobulbar haemorrhage leading to high pressure and postponement of the operation, globe perforation, damage to the optic nerve or ophthalmic artery, or spread of local anaesthetic to the brainstem along the optic nerve dural sheath with the risk of unconsciousness and apnoea.⁶ The patient's medical condition may deteriorate during the procedure.

An increasing number of anaesthetists are now becoming interested and perform the blocks themselves, using either the retrobulbar or more recently peribulbar method.^{7,8} The advantage is that the anaesthetist is involved in the choice of anaesthetic method, the preparation and counselling, monitoring during the procedure and is available to look after the patient's complete welfare and to treat any complications that might arise. Time can be allowed more easily to ensure

adequacy of the block, which includes not only anaesthesia and total immobility of the globe and peri-orbital muscles but also, uniquely, a low pressure within the eye. The anaesthetist will improve the turnover, of patients because time is saved, and the number of operations that may be performed safely may be increased. However, since at the present time many operations are carried out in the absence of an anaesthetist, there would be implications for anaesthetic staffing.

Local anaesthesia should be seen as an alternative method for the more fit and healthy and not as a means of operating on those unfit for general anaesthesia. It is a prerequisite for local anaesthesia for cataract surgery that the patients can lie still and fairly flat comfortably, are not dyspnoeic at rest and do not have uncontrolled coughing. They should neither have a medical history that would predicate intensive cardiac monitoring and pharmacological control, nor have significant coagulation defects. It must be possible to communicate with the patient and they must agree to the method after a full explanation of the steps involved. The surgeon may prefer general anaesthesia for exceptionally complex procedures. There will therefore remain some patients for whom a local anaesthetic is contraindicated.

The patients should be fasted, investigated and prepared as fully as for a general anaesthetic although all sedative drugs should be avoided. Intravenous access must be assured and monitoring including electrocardiogram, noninvasive blood pressure measurement and pulse oximetry should always be used.

The principles of 'painless local anaesthesia' should be applied using initially local anaesthetics at body temperature diluted with balanced salt solution and very fine 25-30-gauge needles^{9,10}. The peribulbar technique requires larger volumes of local anaesthetic than the retrobulbar⁷ but up to 15 ml of the most commonly used mixture of 0.75% bupivacaine mixed with equal parts of 2% lignocaine remains well within the recommended maximum doses.

Hyaluronidase and adrenaline are added frequently to improve spread and increase duration respectively.¹¹ Time must be allowed so that supplementary local anaesthetic may be injected until complete akinesia is achieved, and for the application of pressure devices to ensure low intra-ocular pressure. The peribulbar block is relatively safe and most effective; a separate facial nerve block, which is often painful and temporarily disfiguring, is not required.

Sedative drugs are mostly avoided since these elderly patients tolerate the procedure very well. Not more than 1% will require minimal sedation such as midazolam 1 mg. Sedation may lead to airway problems, decreases in oxygen saturation or sudden arousal. The use of

intravenous induction agents to cover the insertion of the block is unnecessary if painless local anaesthetic methods are used.

The patient may have their operation in a day surgery unit and leave within one hour provided suitable arrangements for ophthalmic follow-up are available.

Even traditional surgeons and anaesthetists are impressed by the simplicity, success and excellent operating conditions provided by the local anaesthetic method and are rapidly converted to it. The swing to local anaesthesia will gather momentum and more cataract surgery is likely to be performed in the future under local anaesthesia unless there is a specific indication for general anaesthesia. It would be to the detriment of our patients and specialty if anaesthetists are not involved.

Charing Cross Hospital,
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Effects of famotidine and cimetidine on plasma levels of epidurally administered lignocaine

K. KISHIKAWA, A. NAMIKI, K. MIYASHITA AND K. SAITOH

Summary

The effects of two H_2 -receptor antagonists, famotidine and cimetidine, on the plasma levels of epidurally administered lignocaine were studied. Group A ($n = 20$) received famotidine 20 mg orally the night before surgery and 20 mg intramuscularly 60 minutes before induction of anaesthesia. Group B ($n = 15$) received cimetidine 200 mg orally the night before the surgery and 400 mg orally 60 minutes before the anaesthetic induction. Group C ($n = 20$) received neither famotidine nor cimetidine and served as controls. Twelve millilitres of 2.0% lignocaine with adrenaline 1:200 000 was injected into the epidural space in all patients, after the establishment of general anaesthesia with nitrous oxide, oxygen, and enflurane (0.3–0.5%). The patients who received cimetidine showed significantly higher plasma concentrations of lignocaine compared with either group A or group C at all investigation times ($p < 0.01$). The mean peak plasma concentrations were 2.4 (SEM 0.1), 3.2 (SEM 0.2) and 2.3 (SEM 0.1) $\mu\text{g/ml}$ in group A, B, and C, respectively. This study suggests that famotidine is preferable to cimetidine for control of gastric acidity before the use of lignocaine as the epidural anaesthetic.

Key words

Anaesthetic techniques, regional; epidural.
Histamine; cimetidine, famotidine.

Cimetidine, an H_2 -receptor antagonist, was used before anaesthesia to reduce gastric fluid volume and acidity as a prophylaxis against aspiration pneumonitis.¹ However, it inhibits the hepatic elimination of many drugs,^{2,3} and it was reported recently that plasma lignocaine levels during epidural anaesthesia for Caesarean section tend to be higher in patients who have received cimetidine.⁴ On the other hand, famotidine, a new H_2 -receptor antagonist, has a different chemical structure⁵ and, unlike cimetidine, does not interfere with hepatic drug elimination⁶ in inhibiting stimulated gastric acid secretion. Famotidine is reported to be 20 times more potent and 1.3 times longer-lasting than cimetidine as an inhibitor of stimulated gastric acid secretion.^{5,7} Famotidine 20 mg intramuscularly was shown to be effective in increasing the gastric fluid pH and decreasing the volume of the gastric contents before anaesthesia.^{8,9}

The present study investigated the effects of famotidine and cimetidine given before anaesthesia on plasma lignocaine levels after epidural administration.

Methods

Fifty-five ASA 1 patients scheduled for elective subtotal gastrectomy or thoracic surgery were studied. Patients with

hepatic, renal, cardiac disease, or asthma were not included. Informed consent was obtained from each patient and the study was approved by the hospital's ethics committee.

Patients were assigned to one of the following three groups: group A ($n = 20$) received famotidine 20 mg orally the night before the surgery, and again 20 mg intramuscularly 60 minutes before the induction of anaesthesia. Group B ($n = 15$) received cimetidine 200 mg orally the night before the surgery, and again 400 mg orally 60 minutes before the anaesthetic induction. Group C ($n = 20$) received neither famotidine nor cimetidine before operation and served as controls.

All patients were premedicated intramuscularly with atropine 0.01 mg/kg and hydroxyzine 1 mg/kg 30 minutes before induction of anaesthesia. A control blood sample was drawn after the insertion of a radial artery cannula for the collection of blood samples and continuous blood pressure monitoring.

A catheter for a continuous epidural block was inserted at T_{7/8} intervertebral space for abdominal surgery, or at C₇/T₁ for thoracic surgery. Lignocaine 1 ml with adrenaline 1:200 000 was used as a test dose. General anaesthesia was then induced intravenously with thiamylal 5 mg/kg

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Accepted 3 October 1989.

followed by suxamethonium 1 mg/kg to facilitate tracheal intubation. Anaesthesia was maintained with nitrous oxide (4 litres/minute), oxygen (2 litres/minute), and enflurane (0.3–0.5%). Twelve millilitres of 2.0% lignocaine with adrenaline 1:200 000 was injected into the epidural space after the establishment of general anaesthesia. The mean arterial blood pressure was maintained above 80 mmHg throughout the investigation period using a vasopressor (ephedrine) whenever necessary. Arterial blood samples for the measurement of plasma lignocaine levels were collected at 10, 15, 20, 30, 40, and 50 minutes after the first epidural injection of lignocaine. All samples were immediately centrifuged and frozen until required for the assay. Arterial blood gas analysis was performed 20 minutes after the lignocaine administration. The plasma levels of lignocaine were analysed by a fluorescence polarization immunoassay (TDX system, Abbot K.K.).

The plasma concentrations of lignocaine were analysed for statistical significance using ANOVA followed by Duncan's test for multiple comparisons, and $p < 0.01$ was considered significant.

Results

There were no significant differences among the three groups with respect to age, sex distribution, body weight, or in their total plasma protein values before the operation, or their arterial blood pH values 20 minutes after the epidural block (Table 1). The number of patients who received cervical or thoracic epidural anaesthesia are also listed in Table 1. No significant differences were observed in the three groups with respect to their mean arterial blood pressure during the investigation period.

The patients who received cimetidine (group B) showed significantly higher plasma lignocaine levels than did those in either group A or C at all times ($p < 0.01$, Fig. 1). The mean peak plasma concentrations of lignocaine were 2.4 (SEM 0.1), 3.2 (SEM 0.2) and 2.3 (SEM 0.1) $\mu\text{g/ml}$ in groups A, B, and C, respectively and the peak plasma concentration occurred 10 minutes after the lignocaine administration in each group.

No side effects were seen that were attributable to either famotidine or cimetidine.

Discussion

The results of this study show that the cimetidine premedicated group had significantly higher plasma lignocaine

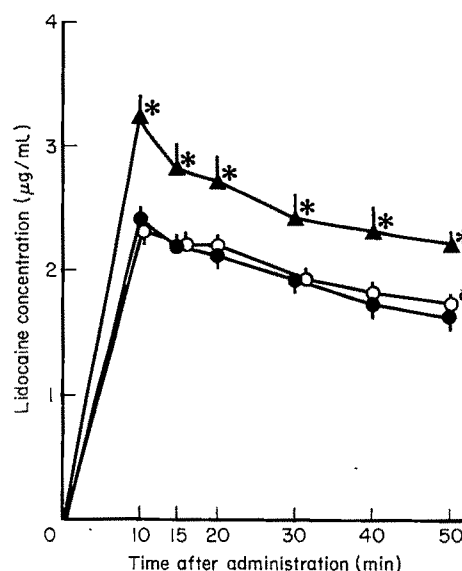


Fig. 1. Mean plasma concentrations of epidurally administered lignocaine in the three groups. * $p < 0.01$, as compared with the famotidine or control group. Bars represent SEM. ●, group A (famotidine); ▲, group B (cimetidine); ○, group C (control).

levels than those who received famotidine or no H_2 receptor antagonist.

The mechanism of the inhibition of lignocaine metabolism by cimetidine remains controversial,¹⁰ although a decrease in hepatic blood flow or an interference with microsomal oxidation in the liver were suggested.^{1–3} In contrast, the clearance of antipyrine, which was generally accepted as an index of the hepatic cytochrome P450 enzyme activity, is not impaired by famotidine in healthy volunteers.¹¹ Furthermore it has been shown that famotidine does not bind to the cytochrome P450 system *in vitro*.⁶ Our results confirm that famotidine has no effects on hepatic drug elimination. It is thus suggested that this new H_2 -receptor antagonist can be used more safely than cimetidine in critically ill patients with liver dysfunction and impaired drug metabolism.

It should be noted in this study that there was a slight difference in the site of the epidural catheter insertion among the groups. However, Mayumi *et al.*¹² demonstrated that there are no differences in the rate of vascular absorption of lignocaine from different parts of the epidural space. Therefore, we consider that the difference in the site of epidural injections did not affect the plasma concentrations of lignocaine.

Ranitidine, an H_2 -blocker, has been also widely used for antacid prophylaxis.¹³ Interactions of ranitidine with simultaneously administered drugs were reviewed by Kirch *et al.*¹⁴ Despite earlier reports indicating that ranitidine did not affect drug metabolism,^{15,16} several recent investigations demonstrated that ranitidine can also cause interactions with some drugs because of its influence on cytochrome P450, but to a much lesser degree than cimetidine.^{17–20} Dailey *et al.*⁴ showed no significant effect of ranitidine on the disposition of epidurally administered lignocaine, but others have demonstrated that patients pretreated with ranitidine had higher plasma bupivacaine concentrations than the control group when bupivacaine was administered epidurally.²¹ Further study of the effects of famotidine on bupivacaine disposition are required because ranitidine

Table 1. Patient characteristics, total plasma (TP) protein before the operation, the number of patients who received a cervical or thoracic epidural administration of lignocaine, and the arterial blood pH 20 minutes after the epidural injection. Values expressed as mean (SEM).

	Group A (n = 20)	Group B (n = 15)	Group C (n = 20)
Age, years	51.8 (2.9)	57.0 (3.0)	52.6 (2.6)
Males/females	9/11	7/8	9/11
Body weight, kg	61.5 (2.8)	57.8 (3.4)	62.4 (2.5)
Height, cm	160.0 (1.2)	161.5 (2.7)	160.1 (2.4)
TP, g/dlitre	7.1 (0.1)	7.2 (0.1)	7.1 (0.1)
Arterial pH	7.42 (0.00)	7.43 (0.01)	7.43 (0.00)
Epidural block			
cervical/thoracic	3/17	5/10	3/17

showed significant effects on bupivacaine metabolism, but not on lignocaine.

Cimetidine premedication was found to have a significant influence on the plasma levels of epidurally administered lignocaine which was not seen with famotidine. The latter drug is therefore to be preferred for aspiration prophylaxis if lignocaine is to be used epidurally.

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Epidurography in premature infants

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Summary

A caudal epidural catheter was inserted in 20 premature, high risk infants for abdominal or thoracic surgery under combined caudal epidural and general anaesthesia. Epidurography was used to confirm the position of the catheter which was found to be misplaced in three patients. The catheter penetrated the dura in one case, in another the tip was located in an epidural vessel and in the third the catheter was seen to be curled up within the epidural space. It was concluded that epidurographic control is essential with this method of anaesthesia in very small infants, in whom it was found to provide considerable advantages despite serious risks.

Key words

Anaesthesia; paediatric.

Anaesthetic techniques; epidural, caudal.

Epidural anaesthesia with the use of a catheter is described for use in children.¹⁻⁴ However, it seems to be employed seldom in premature infants, probably because of technical problems, which include those of access, and fear of the toxic effects of local anaesthetic drugs because of reduced plasma protein binding and prolonged elimination in the very young.

A caudal single shot is the most frequently used regional technique for surgery below the level of the diaphragm in children.⁵ A cranially directed catheter can be introduced via the sacral hiatus in infants to obtain analgesia for upper abdominal and thoracic surgery and can be left in place for postoperative pain relief.⁶ Epidurography can be used to confirm the injection site and the position of the epidural catheter.⁶⁻⁸

Patients and methods

The trial was approved by the hospital ethics committee. Twenty premature infants scheduled for abdominal or thoracic surgery between October 1988 and June 1989 were studied. The surgical indications are listed in Table 1.

All the infants were fasted for 3 hours before surgery. Premedication was with intramuscular atropine 0.01 mg/kg 30 minutes before induction of anaesthesia. Electrodes for the electrocardiograph and probes for plethysmography and pulse oximetry, and an automatic blood pressure cuff,

were attached on arrival in the operating room. General anaesthesia was induced with oxygen/room air and isoflurane (maximum 2%) (in order to avoid intestinal distension by nitrous oxide) using a facemask or tracheal tube previously inserted in the neonatal intensive care unit. An infusion of compound sodium lactate or glucose/saline was begun at a rate of 10 ml/kg/hour. Thereafter vecuronium 0.1 mg/kg was administered to facilitate tracheal intubation (if not already done) and mechanical ventilation of the lungs, by means of a Servo 900B ventilator, and anaesthesia was maintained with a mixture of oxygen/compressed medical air and isoflurane 0.3%. End-tidal carbon dioxide was continuously monitored by a capnograph (Mijnhardt side-stream). Body temperature was recorded by an oesophageal probe.

The infant was then placed in the lateral position. The skin over the sacral hiatus was punctured under sterile conditions with an 18-G intravenous needle before the caudal insertion of a 19-G Tuohy needle (Portex). The patient was tilted into a 20° head down position and after an aspiration test bupivacaine plain 0.25%, 1.25 ml/kg, was injected slowly into the caudal space. A 23-G catheter was directed cranially, through the Tuohy needle until the spinal cord segments to be blocked were reached, as indicated by the marks on the catheter. An X ray was taken using iohexol 300 mg/ml (Omnipaque 300, Nycomed) in normal saline (1:1) as contrast material (total volume 0.5 ml/kg), to check the position of the epidural catheter.

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Accepted 31 January 1990.

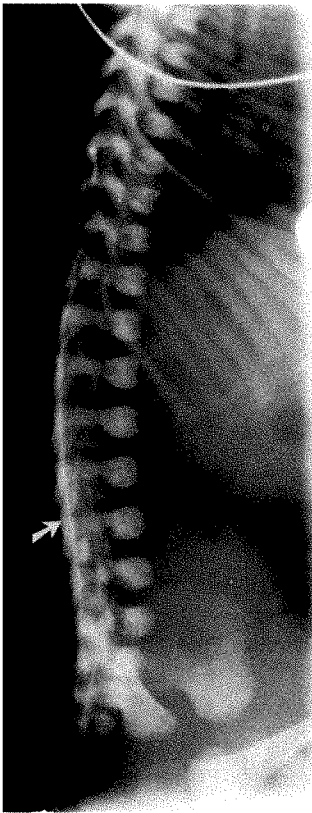


Fig. 1. The arrow shows contrast in the epidural space (lateral view).

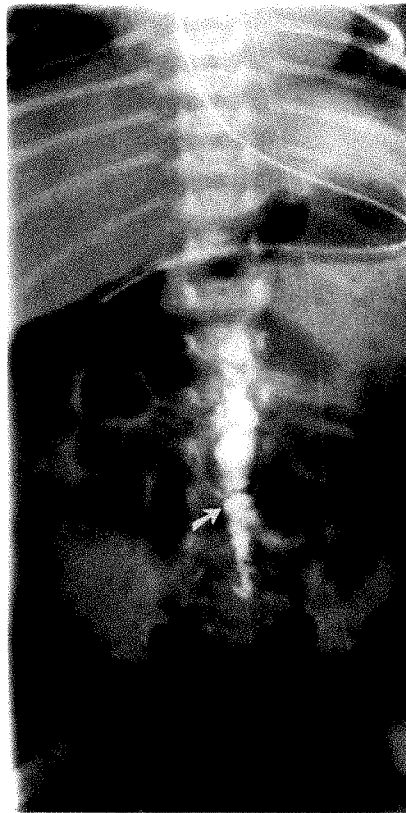


Fig. 2. The arrow indicates the lobulated appearance of the epidural space in the supine position.

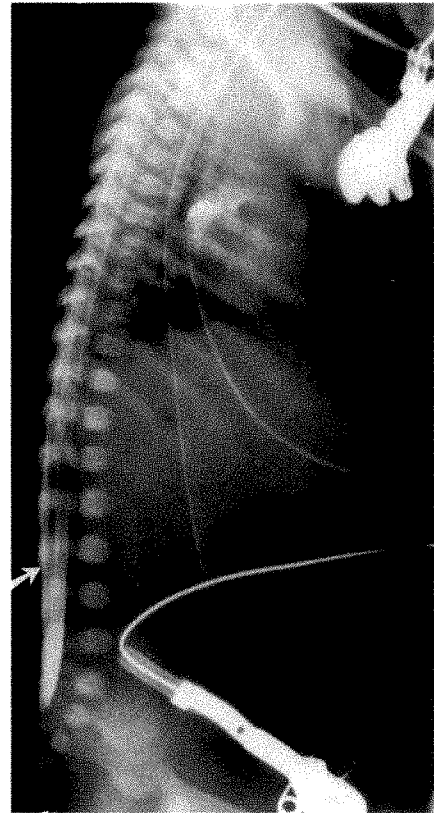


Fig. 3. The arrow indicates a myelogram.

Results

The average age of the 20 infants was 10 days (range 0 to 50). They weighed an average of 1980 g (range 520 to 2750 gram). The various surgical conditions to be treated are listed in Table 1. All infants were described as poor risk.

Eighteen premature infants received a single-bolus caudal block; the 23-G catheter passed easily in a cranial

direction to the required level as determined by the markings on the catheter. There was a slight resistance to the passage of the catheter throughout its introduction. Two patients (5 and 11) for oesophageal surgery received 0.25% bupivacaine 1 ml/kg through the catheter without the bolus caudal injection. Analgesia was considered to be adequate in all the cases since the heart rate did not increase more than 10 beats per minute nor the blood

Table 1. Clinical data.

Patient number	Weight (g)	Age (days)	Diagnosis	Postoperative mechanical ventilation
1	1850	60	Bilateral inguinal hernia	-
2	2300	7	Meconium ileus	-
3	2400	14	Hydronephrosis	-
4	2000	14	Annular pancreas	+
5	2000	1	Oesophageal atresia	+
6	1970	1	Duodenal atresia	-
7	2400	21	Hydronephrosis	-
8	2650	1	Volvulus	-
9	2750	7	Congenital megacolon	-
10	950	1	Duodenal atresia	+
11	2000	1	Oesophageal atresia	+
12	1500	14	Meconium ileus	+
13	2000	14	Necrotising enterocolitis	+
14	2500	24	Necrotising enterocolitis	+
15	1900	7	<i>Extrophia vesicae</i>	-
16	2450	7	Duodenal atresia	+
17	2500	3	Diaphragmatic hernia	+
18	520	3	Hepatic haematoma	+
19	2000	4	Volvulus	-
20	1100	2	Hydronephrosis	+



Fig. 4. Visualisation of epidural vessels (arrows).

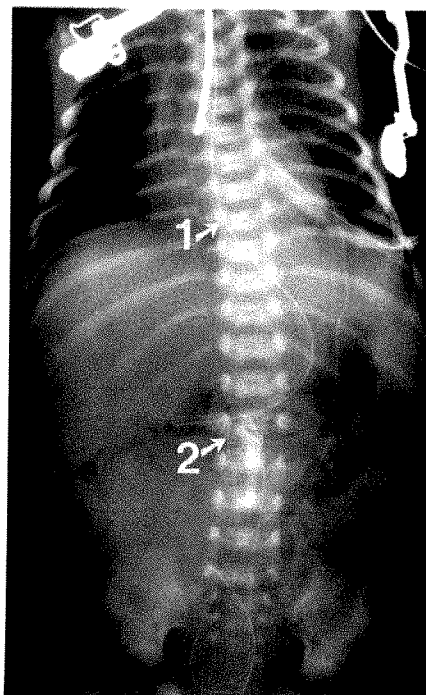


Fig. 5. Arrow 1 shows nasogastric tube, Arrow 2 the curling up of the catheter at T₁₂.

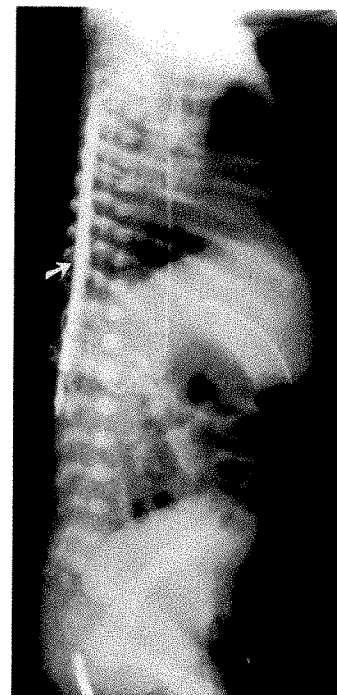


Fig. 6. The arrow indicates epidural contrast (0.125 ml iohexol 300 + 0.125 ml saline) in a 520-g male.

pressure by more than 10 mmHg at the time of the incision, and the capnogram and plethysmogram were unchanged. Surgery lasted between 2 and 2.5 hours and no supplementary dosage of bupivacaine was required.

A catheter was left in place in the 17 infants who had normal X ray findings. Pain relief was achieved for 48 hours with nicomorphine with an infusion pump. The catheter was removed in those infants with abnormal radiological findings and the surgical procedure was performed with the help of the bolus caudal block supplemented with general anaesthesia (case numbers 10, 11 and 16). There were 11 infants who needed mechanical ventilation of their lungs before operation and also after operation. This was because of surgical complications or poor respiratory function. The remaining nine infants' tracheas were extubated at the end of the surgical procedure.

Radiological findings

An X ray was taken in the lateral position and displayed a typical strip of contrast in the spinal canal in seven cases. They confirmed that the tip of the catheter was in the epidural space (Fig. 1). The epidural contrast looked lobulated when the infant was in the supine position (Fig. 2). There were 10 patients in this group. The contrast is also seen to be unilateral. This occurs when the catheter is not in the midline and is due to the viscosity of the substance which delays the spread at first.

No resistance at all was felt on introduction of the catheter in infant number 10. The X ray revealed a myelogram (Fig. 3) although cerebrospinal fluid was not obtained after repeated aspiration. This is probably a result of the small diameter of the catheter (0.63 mm). It was impossible to introduce the catheter further than 2 cm in infant number 12. We were unable to aspirate blood even though

the tip was located in an epidural vessel (Fig. 4). The resistance while introducing the catheter was greater than normal in one case, number 16, and the catheter was seen to be curled up in the epidural space (Fig. 5). Figure 6 shows that an epidural catheter can be inserted in an infant weighing as little as 520 grams.¹

Discussion

Premature infants who have surgery display marked increases in heart rate and blood pressure.^{9,10} Hormonal studies in this age group show an increase in blood levels of stress hormones, during and after operation comparable with, or in excess of, those found in adults, under different types of general anaesthesia.¹¹ Surgical stress results in a postoperative catabolic response which can be both prolonged and considerable.¹¹ Furthermore, psychological sequelae of neonatal pain are postulated. Pain after operations might be expected to exacerbate these undesirable effects.

Booker¹² states that a recent survey showed that 48% of anaesthetists fail to provide any form of analgesia after major surgery in neonates. The reasons for this are probably historical: the discredited idea that neonates do not feel pain and the fear of respiratory depression resulting from the use of narcotic analgesics. Recent work has rendered such an approach untenable and indicates that the increasing use of regional anaesthesia in paediatrics¹³ should extend to the preterm neonate.

Another factor of great importance is the iatrogenic morbidity associated with mechanical ventilation of the lungs in the premature infant.¹⁴ Eleven patients in our series were already receiving mechanical ventilation of the lungs before surgery, but the remaining nine infants were on the verge of respiratory failure and would have undoubtedly

required ventilation after operation if a regional technique were not used. Single shot caudal or spinal anaesthesia are the only other forms of regional block applicable in these tiny patients. The duration of the block cannot be extended with either method longer than that achieved by the addition of adrenaline, and prolonged postoperative analgesia is not provided. Single shot caudal anaesthesia does not extend high enough for operations above the diaphragm.

We injected bupivacaine via the epidural catheter to obtain thoracic anaesthesia in two cases and used this catheter to obtain postoperative analgesia in 17 patients. We used a single caudal shot of bupivacaine for subdiaphragmatic operations, but injection through the catheter would have reduced the volume of drug required. We intend to use the latter method in all cases.

None of our patients required a supplementary dose of bupivacaine, but the majority of operations were of a duration close to that of the maximal duration of action of bupivacaine.

We consider misplacement of the catheter in 15% of our 20 patients to be too high. Bösenberg⁶ used a caudal epidural catheter for biliary surgery in 20 infants and only one failed to reach the required spinal segment. However, his smallest infant was equal in weight to the largest in our series. The authors have considerable experience in the insertion of caudal epidural catheters in full term neonates and infants but this series is a report of the results with their first 20 *preterm* neonates. It was expected that experience would lead to a higher success rate and since this article was written we have used a caudal epidural catheter correctly in a further 10 similar infants.

The advantages of the technique are clear. Our aim in this report, of our very first series, is to indicate the difficulties we encountered and the experience required in the insertion of caudal epidural catheters. Serious complications can be avoided by identification of misplacement by means of epidurography.

Acknowledgment

We thank Miss S. Rijken for secretarial assistance.

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Anaesthetic problems in *ex situ* resection of the liver

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Summary

Ex-situ resection of the liver is a new surgical technique for treatment of liver tumours not amenable to conventional surgery. This paper describes the cardiovascular and metabolic changes that occurred in nine consecutive such patients. No severe haemodynamic or pulmonary complications occurred. Specific problems were encountered during the prolonged anhepatic period, which lasted an average of 5.96 (SD 1.46) hours. Significant metabolic and coagulation disorders occurred 2 to 3 hours after hepatectomy because of complete loss of hepatic function. The predominant findings during the anhepatic period were hypoglycaemia and severe metabolic acidosis, mainly from increased levels of lactic acid. Exogenous administration of dextrose 5% at an average rate of 188 ml/hour was necessary to maintain normoglycaemia, while correction of metabolic acidosis required 403 (SD 159.79) mmol of sodium bicarbonate, supplemented by hyperventilation. Tris-hydroxymethylaminomethane was used when sodium overload was thought to be a problem. There was a marked decrease of Factor V and fibrinogen, a moderate thrombocytopenia and fibrinolysis. The severity of these alterations was dependent on the duration of the anhepatic period and the primary function of the re-implanted liver.

Key words

Anaesthesia; liver transplantation.

Complications.

Ex situ resection of the liver is a new surgical procedure for patients with conventionally unresectable hepatic tumours and with contraindications to liver transplantation. The first such resection was performed in 1988 in a 40-year-old patient suffering from a large metastasis from a leiomyosarcoma and which was regarded conventionally as unresectable.¹ Only liver segments I and IV could be preserved after removal of the 4.5-kg tumour and the surrounding liver tissue, but postoperative liver function was good and the course uncomplicated. The same procedure has been performed since then in eight other patients. The aim in developing the new *ex situ* approach was to increase the resectability rate in patients with advanced tumours, to improve the extent of tumour resection, to avoid liver transplantation in patients with tumours that generally show unsatisfactory results and in patients with benign tumours.² The operation did not extend beyond an exploratory laparotomy if *ex situ* surgery was thought to be impossible.

The major problems for the anaesthetist in this type of surgery is the management of the anhepatic period which lasts several hours and is much longer than during liver transplantation.

This paper describes our experience with these patients.

Methods

Nine patients (five males) are described whose ages range from 30 to 62 years (mean 49.5 years). The indications for surgery were hepatic metastases (four patients), common bile duct tumours with infiltration of the liver parenchyma or the confluence of the hepatic veins (four patients), while one patient had a benign tumour with severe inferior vena cava compression that had caused orthostatic collapse. Six patients are still alive; five were discharged from hospital; three patients died several weeks after the operation.

Technical details

The technique of liver *ex situ* resection and re-implantation is based on that of liver transplantation and was recently described in detail.² The anhepatic phase lasts for several hours, so venovenous (femoro-porto-axillary) bypass with heparin-bonded Gott shunts and a biopump is required.^{3,4} Protection of the liver from ischaemic damage is provided by hypothermic perfusion initially with 6 litres of cardioplegic solution HTK⁵ into the portal vein and 0.5 litres into the hepatic artery.

During the resection the liver is placed in a cold solution

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Accepted 19 February 1990.

and perfusion is repeated at hourly intervals. HTK solution was used in all cases. The suitability of this solution for organ preservation in human liver transplantation has been verified.⁶ This solution seems to be of advantage in this procedure because of its high buffering capacity and even slight warming will not be harmful to the liver. Also, its potassium concentration is low in comparison to other solutions currently used, such as Eurocollins or UW (University of Wisconsin) and consequently it is not as dangerous if it accidentally enters the circulation.

Anaesthesia

The anaesthetic technique and monitoring is as used for orthotopic liver transplantation.⁷ Induction of anaesthesia was achieved with fentanyl 200–300 µg, followed by 5–8 mg/kg thiopentone; suxamethonium 1.5 mg/kg was given to facilitate tracheal intubation with a soft-cuffed tube. Anaesthesia is maintained with isoflurane in a 40% oxygen–air mixture and pancuronium is used as the muscle relaxant. Additional fentanyl was given as required for analgesia. The minute ventilation is adjusted to produce a P_{aCO_2} of 4.2–4.6 kPa. Ringer's solution was infused for fluid replacement. All patients received dopamine prophylactically in a dose of 2–5 µg/kg/minute to prevent renal impairment associated with extensive blood loss and the extended anhepatic period.

Blood loss was replaced with packed red blood cells. In addition, a Haemonetics Cell Saver 4 was used after re-implantation of the liver. Intra-operative coagulopathy was corrected by fresh frozen plasma (FFP) at an approximate ratio to homologous and cell saved red cells of 1:1, while platelets, fibrinogen and factor concentrates were given depending on laboratory results and thrombelastography (TEG).

Intra-operative monitoring

Cardiovascular monitoring consisted of an ECG, direct arterial pressure measurements and a pulmonary artery catheter (Swan-Ganz) for continuous monitoring of central venous pressure (CVP), pulmonary artery pressure (PAP), intermittent measurement of pulmonary artery wedge pressure (PAWP) and thermodilution cardiac output.

End-tidal carbon dioxide concentration was measured with an infrared analyser to adjust the ventilation volume and used as a sensitive indicator of acute reduction in pulmonary perfusion (e.g. air embolism) and sudden changes in cardiac output. Body temperature was monitored by oesophageal and rectal thermometers.

Blood samples

Blood samples were collected at intervals during the various phases of surgery for measurements of blood gases, acid base balance, haematocrit, coagulation profile, electrolytes, lactate and glucose. Coagulation variables included prothrombin time (PT), activated thromboplastin time (PTT), fibrinogen, Factor II, Factor V levels and platelet count.

Thrombelastography (TEG) was recorded in four patients. The thrombelastograph (Thrombelastograph D, Hellige, W. Germany) is a mechanical device that monitors the entire process of coagulation, including the initial

fluid state without fibrin strands, the gradual increase in strength of fibrin (coagulation) and the resolution of fibrin strands (fibrinolysis).⁸ Its simplicity makes it ideal for use in the operation theatre. The variables measured by TEG were reaction time, coagulation time, maximum amplitude, amplitude 60 minutes after maximum amplitude and the whole blood clot lysis index. Normal ranges of these variables are 7.5–15.0 minutes, 2.5–5.0 minutes, 47–67 mm and 100–85% respectively. Prolongation of the reaction time reflects plasma factor deficiency and a markedly prolonged reaction time is seen with heparinisation. A narrowed maximum amplitude indicates platelet and fibrinogen deficiency and clot lysis index below 85% is a sign for fibrinolysis.

Measurements were made after induction of anaesthesia (A in the figures), at the end of the dissection period and before venovenous bypass (B), after hepatectomy (C), at the end of the anhepatic period (D), after re-implantation of the liver (E) and with closure of the abdominal wall (F).

Student's *t*-test was used for statistical analysis with the prerequisite of a nonsignificant test of variance (*F*-test). Differences from pre-operative values with $p < 0.05$ were considered statistically significant (* $p < 0.05$, ** $p < 0.005$, *** $p < 0.0005$ in the figures). All data are presented as means (SD).

Results

The operation time ranged from 9.5–18.0 hours with a mean of 14 (3.0) hours. The average number of units of red cells used was 15.7 (6.78) units, with a maximum of 26 units and a minimum of 4 units. Other replacement consisted of 3.9 (4.4) units of autologous salvaged packed cells, 19 (4.65) units of platelet concentrates and 23 (11.49) units of FFP. Five patients received fibrinogen and prothrombin factor concentrates.

Haemodynamics

The heart rate and mean arterial pressure changes during the entire procedure were unremarkable (Fig. 1). The cardiac filling pressures remained within the normal range and there were no signs of myocardial impairment. There was a gradual increase in CVP from 6.25 (2.38) mmHg at the start of surgery to 9.87 (3.60) mmHg after re-implantation of the liver while the PAWP increased from 7.6 (2.72) to 13

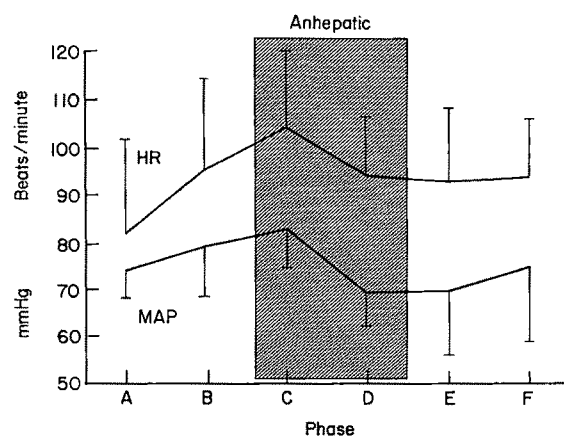


Fig. 1. Changes in heart rate (HR) and mean arterial pressure (MAP) at the various phases during *ex situ* resection of the liver.

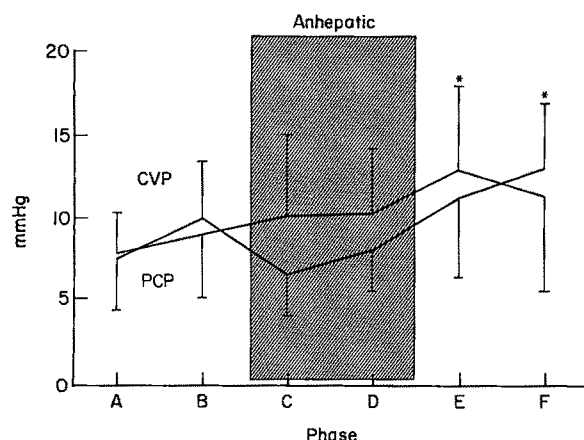


Fig. 2. Changes in central venous pressure (CVP) and pulmonary artery wedge pressure (PCP) during *ex situ* resection of the liver.

(3.88) mmHg at the end of surgery (Fig. 2). Systemic vascular resistance increased from 965.6 (200.58) to 1177.6 (342.88) dynes.second/cm⁵ at the time of hepatectomy but then decreased during the anhepatic stage to a minimum after reperfusion and was 755.33 (297.97) dynes.second/cm⁵ at the end of surgery. The alterations were not significant compared to the pre-operative values. The cardiac index remained constant during the entire bypass period with only a moderate increase after revascularisation of the liver (Fig. 3).

In contrast the stroke volume index showed a continuous reduction until post hepatectomy from 44.2 (9.23)–30.56 (11.11) ml/sq m, but values returned to baseline levels after reperfusion. The left cardiac work index remained relatively constant and showed good left ventricular function in all the patients (Fig. 4).

Renal function

No signs of renal impairment were observed in any patient. The average urine output was between 105 and 517 ml/hour. Apart from the routine use of dopamine three patients required a single bolus of frusemide to maintain urine output above 100 ml/hour.

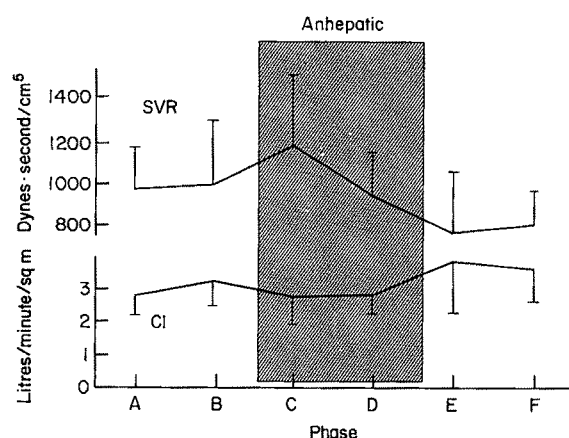


Fig. 3. Changes in systemic vascular resistance (SVR) and cardiac index (CI) during *ex situ* resection of the liver.

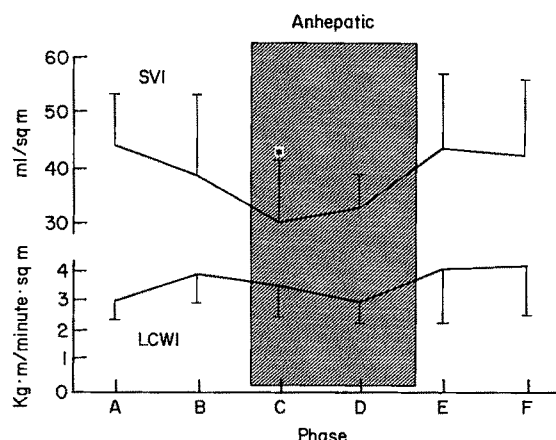


Fig. 4. Changes in stroke volume index (SVI) and left cardiac work index (LCWI) during *ex situ* resection of the liver.

Metabolic changes

The most striking findings were severe metabolic acidosis and a significant elevation of blood lactate levels. The arterial blood pH declined progressively from 7.47 (0.04) to 7.25 (0.09) after reperfusion, although acidosis was treated continuously by an average supplementation of 403.33 (159.79) mmol of sodium bicarbonate and 31.11 (45.07) ml of tris-hydroxymethyl aminomethane (THAM).

The arterial base excess decreased at the same time from -0.5 (1.57) to -7.88 (6.5) mmol/litre. Lactate levels progressively increased from 1.11 (0.47) to 13.67 (4.77) mmol/litre during the procedure (Fig. 5).

The serum electrolytes remained relatively stable despite the severe acid base disturbances (Fig. 6). Levels of sodium gradually increased during the anhepatic period as a result of infused sodium bicarbonate from 142.71 (1.82) to 150.25 (6.26) mmol/litre and decreased slightly towards the end of surgery. Serum potassium remained within normal range; hyperkalaemia after reperfusion was not observed (Fig. 6). One reason for this might be the fact that we were using HTK solution which contains less potassium than other preserving solutions.⁹

Another predominant finding during the anhepatic stage was the development of hypoglycaemia; in one patient the

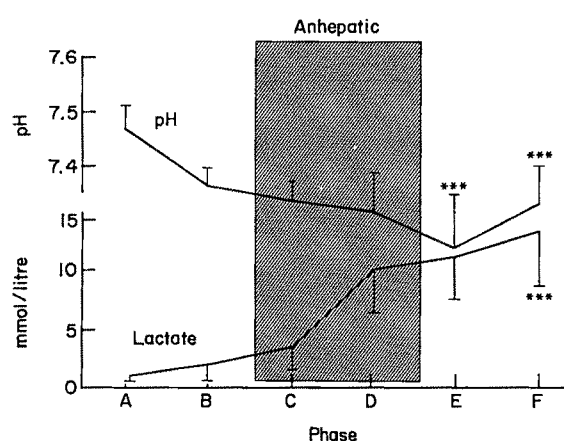


Fig. 5. Changes in arterial plasma hydrogen ion concentration (pH) and lactate levels during *ex situ* resection of the liver.

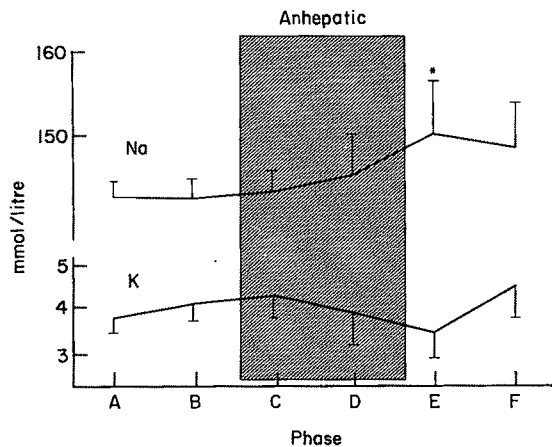


Fig. 6. Changes in levels of serum potassium and sodium during the *ex situ* resection of the liver.

blood sugar decreased from 7.3 to 1.6 mmol/litre during the anhepatic phase. Therefore our patients routinely received an infusion of dextrose 5% with the result that blood glucose levels showed a continuous increase with a peak of 15.7 (4.82) mmol/litre at the end of surgery (Fig. 7). Such therapy is not required during liver transplantation in our unit.

Body temperature

Another problem of *ex situ* liver resection is the development of hypothermia mainly caused by several hours of extracorporeal venovenous bypass and the *in situ* perfusion of the liver with 8 litres of cold protective solution. A thermally controlled blanket and a heating device for blood transfusion were used, but the initial mean oesophageal temperature of 36.0°C decreased to 33.93 (0.96)°C at the end of surgery (Fig. 8).

Coagulation profile

There was a moderate increase of PTT from 38.6 (8.33) to 64.14 (52.16) seconds, a mean decrease in fibrinogen levels of 42.5% and a significant fall in mean platelet counts of 61% with a minimum after reperfusion (Fig. 9).

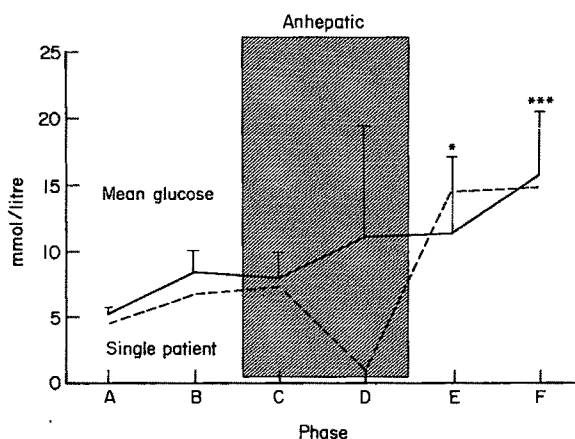


Fig. 7. Blood glucose levels in a single patient without administration of dextrose 5% during the anhepatic period of *ex situ* resection of the liver, and mean values of nine patients.

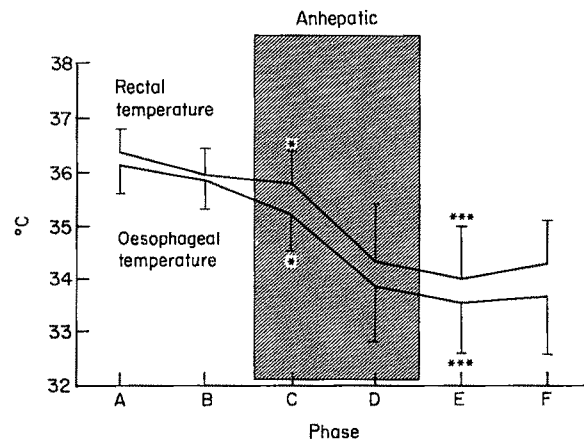


Fig. 8. Changes in oesophageal and rectal temperature during *ex situ* resection of the liver.

The most striking alteration was related to Factor V, with a highly significant decrease from 100% to 46.38 (19.99)% after re-implantation of the liver. This occurred despite administration of FFP (Fig. 10).

In order to illustrate the different findings in coagulation profiles, two patients monitored by TEG are presented. In the first, the TEG was normal until the end of Stage I, but 4.5 hours later during the anhepatic period fibrinolysis rapidly developed. Twenty minutes after reperfusion clot formation stopped completely and the TEG plot was a straight line. This observed coagulation defect coincided with increased oozing in a previously dry surgical field. Transfusions of FFP, platelets and fibrinogen were able to return the TEG pattern to normal by the end of surgery (Fig. 11). In the second patient the infusion rate of FFP was increased prophylactically after hepatectomy. Even though the anhepatic period lasted 9 hours the TEG patterns remained normal during this entire period. However, 2 hours after reperfusion, due to massive haemorrhage combined with technical problems with the portal vein anastomosis and primary failure of the re-implanted liver, the reaction time was prolonged and the maximum amplitude decreased continuously. Even massive transfusion of FFP, platelets, factor concentrates and fibrinogen was not able to reverse this course and due to the poor liver function the TEG patterns did not improve (Fig. 12).

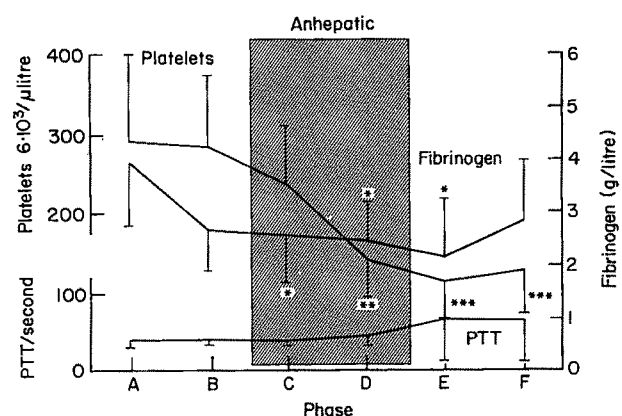


Fig. 9. Changes in activated thromboplastin time (PTT), levels of fibrinogen and platelet counts during *ex situ* resection of the liver.

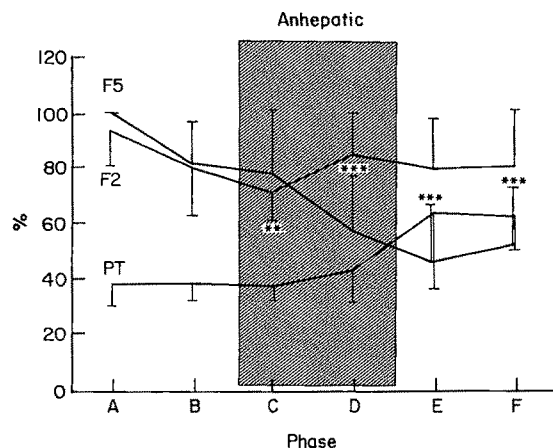


Fig. 10. Changes in prothrombin time (PT) and levels of factor II and V during *ex situ* resection of the liver.

Discussion

Ex situ resection of the liver is a new challenge even for anaesthetists experienced in orthotopic liver transplantation. Specific features of this new surgical procedure are the prolonged anhepatic period, with resultant complete loss of functional and metabolic capacity of the liver as well as the risk of acute liver failure after re-implantation, because of the reduction in the amount of liver tissue. At present it is unknown how long a human can remain in an anhepatic state. In some cases, where it has been necessary to perform an acute hepatectomy due to initial nonfunction and necrosis of the liver allograft, an anhepatic period up to 14 hours was tolerated without severe metabolic disturbances, except for an increase in lactate levels.¹⁰

Based on our preliminary experience with *ex situ* resection, one can say that from the anaesthetic point of view

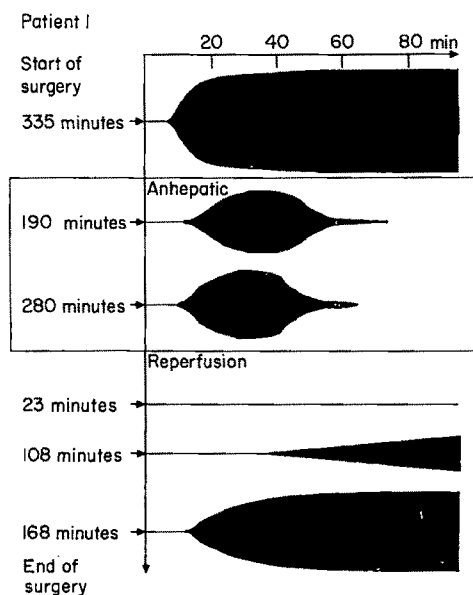


Fig. 11. Thrombelastographic pattern of one patient with severe fibrinolysis during the anhepatic stage and after reperfusion. Selective transfusion of FFP, platelets and factor concentrates normalised TEG pattern at the end of surgery. This patient was transfused 16 units of red cells, 33 units of FFP, 10 units of platelets, 10 g, fibrinogen and 2200 units of prothrombin factor concentrates.

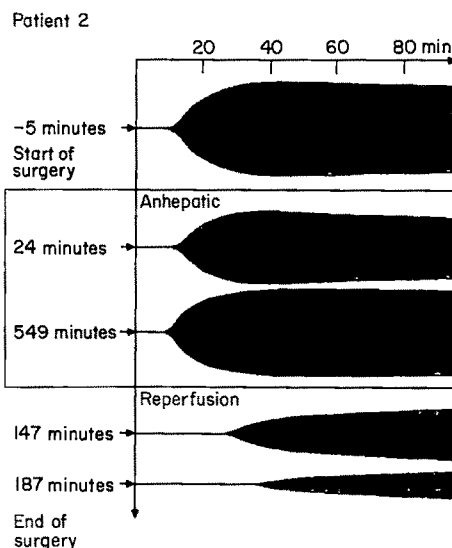


Fig. 12. Thrombelastographic pattern of one patient. Two hours after reperfusion there was continuous prolongation of reaction time and decrease of maximum amplitude due to primary liver failure after revascularisation. This patient was transfused 23 units of red cells, 49 units of FFP, 20 units of platelets, 4 g fibrinogen and 600 units of prothrombin factor concentrates.

the procedure was well tolerated by all nine of our patients. No severe haemodynamic, pulmonary or renal complications occurred during the operation. Details of the surgical procedure, the problems of establishing indications and the results of long-term outcome are published elsewhere.² As with liver transplantation, effective inferior vena caval and portal vein decompression is of major importance in the maintenance of cardiovascular stability and preservation of renal function.^{3,4}

On the other hand, our studies did reveal metabolic and coagulation disorders during the anhepatic period. They are most likely to occur 2–3 hours after hepatectomy. Their severity depends on the duration of the anhepatic period and the function of the liver after re-implantation. The predominant findings were hypoglycaemia and severe metabolic acidosis, mainly because of increased levels of lactate.

Significant changes in blood glucose levels are to be expected during a long anhepatic period because of the major role of the liver in glucose homeostasis. Knowledge about glucose metabolism during orthotopic liver transplantation is still incomplete, although hyperglycaemia is most frequently reported.^{11,12} For this reason exogenous administration of dextrose is currently considered unnecessary during the anhepatic stage of liver transplantation. In contrast, we observed in one of our patients a marked decrease in blood glucose, as low as 1.6 mmol/litre, possibly caused by continuous glucose utilisation and decreased glucose production from reduced gluconeogenesis.¹³ Our other patients were given dextrose 5% at an average of 188 ml/hour during the anhepatic stage to prevent hypoglycaemia. As a result mean values of blood glucose were comparable to those of our patients who had undergone liver transplantation and who received no infusion of dextrose.¹⁴

The cause of metabolic acidosis is reduced metabolism of citrate, lactate and other acids in the absence of hepatic function.¹⁵ The elevated lactate concentrations and the

simultaneous occurrence of hypoglycaemia suggest a decrease in hepatic gluconeogenesis. The severity of the acid-base disturbances and the elevation of lactate levels was dependent on the duration of the anhepatic period as well as on the extent of liver function. Lactic acid concentrations showed a marked individual variability, with the lowest at 5.4 and the highest at 21.6 mmol/litre. The latter was recorded in a patient with an anhepatic time of 9 hours and primary liver failure after revascularisation.

The prolonged anhepatic period during the *ex situ* resection produces marked coagulation disorders as in liver transplantation.¹⁶⁻¹⁷ There was significant consumption of Factor V and fibrinogen, and a moderate thrombocytopenia and fibrinolysis as shown by TEG because of lack of synthesis of coagulation factors and diminished clearance of activated intermediates of fibrinolysis. To clarify the pathological mechanism underlying the observed coagulopathy, further investigations are required using specific fibrinolytic tests. The prolonged bypass period may result in activation of Factor XII on the surface of the bypass system, which will be an additional cause of fibrinolysis.

Our preliminary experience with these patients suggests the following management during the anhepatic phase. Arterial blood gases and blood glucose levels have to be closely monitored in order to prevent hypoglycaemia and severe metabolic acidosis. Dextrose 5% and sodium bicarbonate should routinely be infused during this time. Ringer's lactate solution should be avoided. Hypothermia should be minimised by the use of a thermally controlled blanket and a heating device for inhaled gases and transfused blood. Standard coagulation parameters have a poor correlation to TEG variables. TEG monitoring and TEG guided therapy appears to be of great clinical value in order to correct haemostasis disturbances reliably. By paying attention to these points *ex situ* resection of the liver can be performed without major difficulties.

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Combined air and oxygen entrainment

Effect on the percentage output of fixed performance masks

M. A. LYEW, A. J. HOLLAND AND I. R. METCALF

Summary

Secondary oxygen was added to air entrained by the primary jet of a fixed performance mask. Twenty such masks, in each of six groups rated at 24%, 28%, 31%, 35%, 40% and 50%, gave higher O₂ percentages equivalent to those calculated from an equation that related the O₂ concentration to the secondary flow. Actual values of both were closely correlated, with regression slopes and intercepts similar to those derived from this equation. This technique is simple and predictable in altering the composition of an air–oxygen mixture delivered to the patient.

Key words

Oxygen; delivery systems.

Fixed performance masks use a primary jet of oxygen to entrain room air and produce a gas mixture with a particular oxygen percentage. The inspired oxygen concentration remains fairly steady if the total flow of oxygen-enriched air is greater than the peak inspiratory flow rate of the patient.¹ The peak inspiratory flow has been estimated to be 30 to 35 litres/minute in resting healthy adults.² This flow can be higher during hyperventilation, and, on exceeding the rate of delivery of the gas mixture, result in a decrease in the inspired oxygen concentration.

The entrainment of air occurs in constant proportion to the primary flow, and is dependent on the relative sizes of the oxygen nozzle and entrainment ports.³ These constitute the air–oxygen blender located at the end of the mixing barrel, which conducts the gas mixture into the mask. Nonadjustable masks have a selection of nozzle bores and a common or dissimilar size of entrainment port to give several oxygen concentrations. The aperture of the adjustable masks' entrainment port can be altered to change this concentration.⁴ However, these devices have a limited set of oxygen percentages, which vary in range with the different masks available.

A simple technique is described, which widens the choice of oxygen percentages provided by these types of masks, and retains the advantages of their use. The change in concentration of the delivered gas mixture, as a result of the entrainment of both air and oxygen, was investigated. The clinical application of this method is discussed.

Theory

When air is entrained by a primary jet of oxygen \dot{V}_p , the ratio X of air to oxygen flow is given by:

$$X = \frac{100 - C_n}{C_n - 21} \quad (1)$$

where C_n is the resultant oxygen percentage of the mask, and the concentration of oxygen in room air is taken to be 21%.⁵

If a secondary flow of oxygen \dot{V}_s is entrained with room air \dot{V}_a , the entrainment ratio X is hardly altered, since the densities of air and an air–oxygen mixture are similar. Thus, at the same primary oxygen flow, the entrained flow remains approximately the same i.e.

$$\dot{V}_s + \dot{V}_a = X \cdot \dot{V}_p \quad (2)$$

The new oxygen concentration C in the mask is given by:

$$C = \frac{\text{total O}_2 \text{ flow}}{\text{total gas flow}} = \frac{\dot{V}_p + \dot{V}_s + \dot{V}_a \cdot 21/100}{\dot{V}_p + \dot{V}_s + \dot{V}_a} \times 100 \quad (3)$$

On substituting equation (1) in (2), and (2) in (3), it can be shown that:

$$\frac{\dot{V}_s}{\dot{V}_p} = \frac{C - C_n}{C_n - 21} \quad (4)$$

and that:

$$C = C_n + \frac{C_n - 21}{\dot{V}_p} \cdot \dot{V}_s \quad (5)$$

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Accepted 7 December 1989.

Thus, in principle, at a set primary flow rate, the new oxygen concentration provided by the mask is linearly related to the secondary flow of entrained oxygen. The constant of proportionality is determined in part by the difference between the oxygen concentration of the mask, due only to the primary flow, and that of room air.

Method

The relationships suggested by equation (5) were tested with the apparatus shown in Figure 1. An adult size AirLife Percento mask (American Pharmaseal Company, Valencia, Ca 9135-8900, USA), was strapped on the face of an Ambu resuscitation model. The gas mixture within the mask was sampled with a catheter sited at the nostril and analysed with a Perkin Elmer 1100 MGA mass spectrometer, previously calibrated with air and 100% oxygen. A Portex 600 Thermovent filter was connected to the inlet of the humidity adapter to moderate a secondary flow of oxygen and increase the likelihood of its entrainment with room air. Primary (entraining) and secondary (entrained) oxygen flows were nominally set with Medigas FM pressure-compensated flowmeters, and were determined beforehand with a W.E. Collins spirometer and chart recorder.

Twenty blenders in each of six groups, nominally rated at 24%, 28%, 31%, 35%, 40% and 50%, were randomly chosen for attachment to the mixing barrel. The recommended basal primary flows were used. The mask oxygen concentration was measured before and after the secondary flow was initiated and increased in steps of 1 litre/minute up to a maximal readable setting of 15 litres/minute (measured flow: 15.5 litres/minute). Using least squares regression, the results were compared with those calculated from equation (5). The linearity of the oxygen concentra-

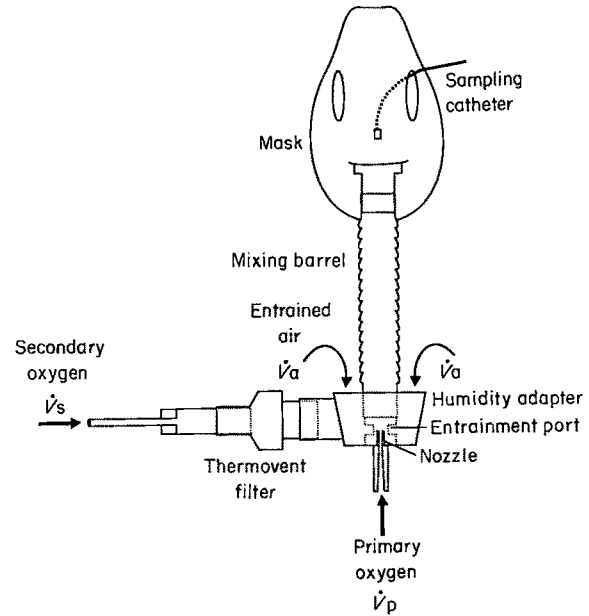


Fig. 1. Mask modified to entrain air and oxygen.

tion as a function of the secondary flow was also statistically investigated, and compared with that of equation (5).

Results

Table 1 shows the nominal and mean actual percentages obtained for each group of blenders, when primary flows were initially used. There was close correlation and agreement between measured and calculated oxygen concentrations over the range of subsequent secondary flows; 95%

Table 1. Concentrations and flows in the masks. Slopes and intercepts derived from equation (5) similar to the concentration-flow regression line data.

Entrainment mask groups						
Concentration (%)						
Nominal	24	28	31	35	40	50
Mean initial (SD)	24.7 (0.57)	27.6 (0.82)	30.8 (1.2)	34.4 (0.98)	39.6 (0.81)	48.9 (0.74)
Primary flow \dot{V}_p (litres/minute)						
Set	2	4	6	8	8	12
Actual	1.94	3.89	5.86	7.84	7.84	11.83
Concentration (SD) (%)	56.50 (5.5)	55.10 (4.0)	57.10 (4.7)	60.90 (3.1)	75.50 (2.3)	83.60 (1.2)
at peak secondary flow \dot{V}_s						
Regression line data						
Actual as compared with predicted concentration (%)						
correlation coefficient	0.99	1.00	1.00	1.00	0.98	0.95
slope	1.04	1.01	1.00	0.99	0.98	0.95
y-intercept (%)	-1.30	-0.32	0.01	0.84	1.20	2.60
95% limits of line (%)	2.60	1.10	1.30	1.30	1.40	1.70
Actual concentration (%) as compared with secondary flow (litres/minute)						
correlation coefficient	0.95	0.95	0.93	0.97	0.99	0.99
slope	1.98	1.74	1.64	1.65	2.39	2.23
y-intercept (%)	24.6	27.7	30.9	34.4	39.8	49.4
95% limits of line (%)	6.6	5.3	6.0	3.9	3.3	2.7
Mean initial O_2 —21%						
Primary flow	1.91	1.70	1.68	1.71	2.37	2.36
Probable error at peak \dot{V}_s of predicted concentration	11.60	5.30	3.66	2.98	4.04	2.92

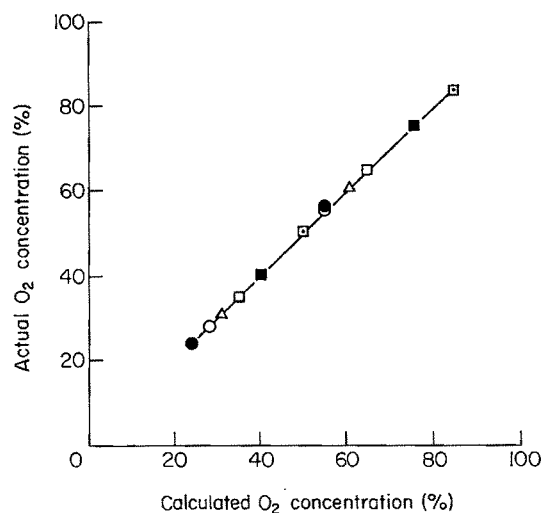


Fig. 2. Measured as compared with calculated oxygen concentration over 0–15 litres/minute secondary flow. Regression lines delimited by symbols for each group of blenders: ●, 24%; ○, 28%; △, 31%; □, 35%; ■, 40%; ▣, 50%.

confidence limits of the regression line were less than 3% (Fig. 2). Average concentrations at 15.5 litres/minute secondary flow were similar for the 24%, 28%, 31% and 35% masks. This was partly because of the increased primary flows recommended for the higher concentration masks to compensate for the decrease in their entrainment ratios and thus maintain similar total flows of the air–oxygen mixtures. Primary flows and initial mask percentages, less the room air oxygen concentration, are opposing factors that determine the slope of equation (5). The slopes became less with masks rated up to 35%. They were greater for the 40% and 50% masks, which therefore showed higher oxygen concentrations at the peak secondary flow.

The correlation between actual mask percentages and secondary oxygen flows was less exact than that between actual and calculated mask concentrations (Fig. 3). This resulted from the variation in actual percentages initially measured in each group with only the primary flows. The effect of secondary flows on oxygen concentration would be decreased if the initial actual value was lower than the

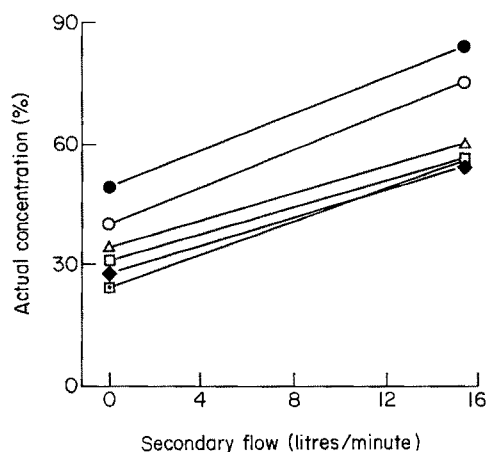


Fig. 3. Measured oxygen concentration as compared with secondary flow over 0–15 litres/minute. Regression lines delimited by symbols for each group of blenders: ●, 24%; ○, 28%; △, 31%; □, 35%; ■, 40%; ▣, 50%.

nominal concentration, and increased if it was higher than that percentage. The slopes of the regression lines were approximately equal to the differences between the mean initial mask concentrations and room air values divided by the primary flows; this supports the validity of equation (5).

The accuracy of the mass spectrometer in determining the oxygen concentration is 0.1%, whereas the flowmeters have a quoted accuracy of 5% of the nominal flow. The probable error of the predicted oxygen percentage can be determined from the principle of superposition of errors.⁶ Differentiation of equation (5) shows that the probable error increases directly with the secondary flow and decreases inversely with the primary flow. Masks rated higher than 24% showed maximal errors at 15.5 litres/minute secondary flow that were similar to those quoted for the Airlife blenders ($\pm 5\%$).

Discussion

The results suggest that delivered concentrations, caused by the entrainment of air and moderate flows of oxygen, are predictable and accurate to a degree similar to that quoted for these masks in conventional use. The initial mask concentration, which results from the entrainment only of air, should be measured with an oxygen analyser, since it may be significantly different from the percentage specified for the mask.⁷ Delivered concentrations are little affected by the face, because the air–oxygen mixture flows freely through the vents of the mask.⁸ The secondary flow of oxygen probably introduces additional turbulence in the mixing barrel, because mask concentrations were found to vary over a 1 to 2% range. Peak values were recorded in our study.

Primary flows, higher than the recommended basal values, may be employed to increase air entrainment and thus total gas flows, so as to reduce dilution with air drawn through the holes of the mask during inspiration. A total flow of up to 60 litres/minute is often recommended for the low volume facemask to lessen the disparity between the delivered and inspired oxygen fractions.⁹ The concentration–flow gradient and thus the maximum oxygen concentration at the peak readable secondary flow would be reduced. In theory, if the secondary flow was equal to the product of the entrainment ratio and the primary flow, 100% oxygen would be supplied by the mask. However, such a flow would be excessive, even if a 50% mask was used.

The modified air entrainment system retains the advantages of high air flow with oxygen enrichment.¹⁰ It avoids the need for a leak-free fit to the patient. A consistent inspired oxygen fraction allows interpretation of changes in arterial P_{O_2} caused by alterations in the ventilatory pattern or pulmonary function. The delivered oxygen concentration can be set with our device by simply adjusting a secondary flow of oxygen in relation to the primary flow. This widens the choice of concentrations available with existing equipment. In fact, only two blenders are needed to provide, respectively, a range of low and high concentrations. Secondary oxygen flows can easily be humidified without affecting the desired oxygen fraction delivered to the mask, if the entrainment ports are not occluded by moisture. High oxygen concentrations are possible without recourse to alternative reservoir systems which have a significant inherent deadspace.

Many patients who require oxygen supplementation are satisfactorily treated with variable performance devices.¹¹ Fixed performance systems are preferred for use in respiratory conditions in which the imprecise administration of oxygen may be hazardous.¹⁰ Oxygen at high concentration can aggravate the instability of lung units caused by an increased ventilation-perfusion mismatch. The effect is opposed by nitrogen inspired at a 30 to 40% concentration, which may act as an 'internal splint' for the air spaces. Thus, 60% has been regarded as the highest consistent oxygen concentration that can safely be inhaled for more than several hours. However, when an F_{IO_2} in excess of 40% is required, simple facemasks with high flows of air and oxygen are often used instead of the air entrainment types.⁹ A separate air supply is unnecessary with our device, which allows a 40 or 50% mask to give a 60% concentration, when the secondary flow is respectively 20/19 or 10/29 of the primary flow (see equation (4)).

The sum of the primary and secondary oxygen flows, estimated to produce a desired percentage, can be shown to be similar to the primary flow of a blender rated at that concentration, when the total flows of the air-oxygen mixtures are kept equivalent. Thus, substantial oxygen flows are still needed to produce adequate total flows of high O_2 concentration. On the other hand, the oxygen percentage of low concentration masks can be cautiously increased with more modest secondary flows. This may be useful in the treatment of patients with an hypoxic respiratory drive who are at risk from CO_2 narcosis. Secondary flows needed to produce a desired oxygen percentage can be estimated from nomograms based on equation (4).

The disadvantage of our system is that it requires two flowmeters for its operation. A proportional unit, similar to the monitored dial mixer, which allows adjustment of the oxygen supply in the ratio of \dot{V}_s/\dot{V}_p and the magnitude of the primary flow, would be ideal for use with this device, since it would eliminate the errors in estimating the concentration due to the reading of separate oxygen flowmeters.

In conclusion, combined air and oxygen entrainment provides a useful method to alter the delivered oxygen percentage, because no adjustment or replacement of the

mask is needed. The effects of mask design and respiratory factors on the clinical performance of this device are minimised by giving adequate total flows of the gas mixture with the required concentration.

Acknowledgments

The authors thank Dr J. Bates and Mr S. Filiatrault of the Meakins-Christie Laboratories, McGill University, for advice and technical assistance. We are also grateful to Mr J. Vézina, chief respiratory technologist, for loan of the equipment, and to Ms C. Colligan for typing the manuscript.

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Autonomic dysfunction and Guillain-Barré syndrome

The use of esmolol in its management

M. A. CALLEJA

Summary

A 17-year-old girl with Guillain-Barré syndrome and autonomic dysfunction was treated successfully with esmolol. Esmolol may be an appropriate drug for the rapid assessment and control of tachyarrhythmias in critically ill patients.

Key words

*Neuropathy; acute inflammatory.
Sympathetic nervous system; beta-adrenergic antagonists, esmolol.*

Acute postinfective polyneuropathy (Guillain-Barré syndrome, GBS) is often complicated by autonomic dysfunction. One such case is described together with suggestions for the management of autonomic dysfunction.

Case history

A 17-year-old girl (weight 50 kg) presented to a district general hospital with a 5-day history of progressive weakness in all four limbs and progressive deterioration of ventilation, phonation and swallowing. Altered sensation (to touch and pinprick) was present in all four limbs with paraesthesiae distally. She described a mild diarrhoeal illness at the onset of her symptoms.

Physical examination showed hypotonia, weakness and areflexia in all limbs. Weakness was assessed as grade 4 on a 5-point scale. Examination of the cranial nerves was normal. Her forced vital capacity (FVC) was 1.4 litres (supine). Arterial P_{O_2} was 13.5 kPa, and P_{CO_2} 5.3 kPa. Cerebrospinal fluid protein concentration was raised at 2.29 g/litre with a markedly elevated globulin fraction. A diagnosis of GBS was made.

She was transferred to our regional neurosciences intensive therapy unit (NITU) the following day. The FVC had decreased to 1 litre and there was increasing difficulty with ventilation, phonation and swallowing. P_{aO_2} was 11 kPa and P_{aCO_2} 6.4 kPa. Artificial ventilation of the lungs was deemed necessary and the trachea was intubated. Sedation and analgesia were achieved with midazolam and morphine by intravenous infusion. Plasmapheresis was performed and repeated on alternate days for 10 days. A tracheostomy was performed 12 days after tracheal intubation.

A tachycardia (140 beats/minute) and pyrexia (38°C) developed 24 hours after admission. Sputum culture suggested a *Staphylococcus aureus* chest infection. This was treated with flucloxacillin. The tachycardia persisted after treatment of the infection. It was thought to be due to autonomic dysfunction. Slowing of the heart rate was noticed in association with vigorous intermittent positive pressure ventilation (IPPV) during physiotherapy. Sweating episodes started 21 days after admission with an increase in heart rate to 180 beats/minute. A recurrent staphylococcal chest infection was treated successfully with no reduction in heart rate or incidence of sweating episodes. Autonomic dysfunction was again thought to be the most likely cause and treatment with esmolol (Brevibloc, Du Pont Pharmaceuticals) was started. A 2.5 mg (50 µg/kg) loading dose was given over one minute, followed by an infusion at 250 µg/minute. A further loading dose of 2.5 mg was given after 4 minutes and the infusion rate was increased to 500 µg/minute. The heart rate decreased to 100 beats/minute, but arterial pressure and urine output remained stable.

The infusion was stopped 8 hours later and the tachycardia returned. Treatment with esmolol was restarted following the above loading dose procedure. Oral treatment with atenolol 50 mg once a day was also started. The esmolol infusion was stopped after 12 hours. The heart rate remained at 110 beats/minute. Treatment with atenolol was continued for the remainder of her stay. No further episodes suggestive of autonomic dysfunction occurred.

The patient was discharged from the NITU after a 70-day stay, which included 65 days of artificial ventilation. Her tracheostomy has since been removed and she is making a good recovery with intensive physiotherapy.

Discussion

Mild autonomic disturbance is reported to occur in 65% of patients with GBS.¹ Sinus tachycardia is one of the most frequent manifestations and hypertension and postural hypotension are common.² Earlier reports suggested a worse prognosis in the presence of autonomic dysfunction,³ but more recent work has refuted this.²

An approach to management that takes autonomic function into account has been suggested.³ GBS can produce an interruption of autonomic reflex pathways in the sympathetic and/or parasympathetic nervous systems.⁴ Pulmonary artery occlusion pressure (PAOP) monitoring has been suggested as an aid to management.⁵ This was not used in this case because of the presence of recurrent staphylococcal infection. Also, PAOP monitoring has not been found necessary in the management of tachycardia.⁵

Excess sympathetic activity is the likeliest cause of this patient's tachycardia and sweating episodes. Insufficient parasympathetic activity is another possibility, although the reduction in heart rate with vigorous IPPV (in effect a normal Valsalva response) does not support this. Beta-blockade was thought desirable to avoid long periods of tachycardia and possible cardiac decompensation. A short-acting beta-blocker was thought necessary to enable the cautious assessment of beta-blockade in the presence of co-existing infection.

Esmolol, a cardioselective beta-blocker, is recommended for use in critically ill patients because of its fast onset and offset of action.⁶ Esterase metabolism accounts for a rapid total body clearance of 285 (ml/kg)/minute and an elimination half-life of 9.2 minutes.⁷ Its esterase-dependent metabolism suggests that it will not accumulate in patients with hepatic or renal impairment. Hypotension is its commonest side effect. This is related directly to the duration of infusion, but frequently resolves during treatment or within 30 minutes of discontinuing the infusion.⁷

Hypotension was not a problem in this patient. Irritation at the intravenous injection site occurs in 10% of patients.

Treatment with a longer-acting beta-blocker was started

after a satisfactory response to betablockade had been obtained with esmolol. This provided reliable control of sympathetic overactivity. A change to an alternative anti-arrhythmic after short-term control has been achieved with esmolol is suggested in the manufacturer's draft data sheet (Du Pont Pharmaceuticals, personal communication). The use of esmolol infusions for longer than 24 hours has not been evaluated thoroughly.

Esmolol provided safe, reliable, rapid control of sympathetic overactivity in this patient, allowing the response to beta-blockade to be assessed. This may be a safer initial approach to betablockade in critically ill patients before treatment with longer-acting, less reversible drugs is started.

Acknowledgment

I thank Dr D.B. Lawrie of Du Pont Pharmaceuticals for his assistance.

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Loss of consciousness after emergence from anaesthesia

A case of suspected micturition syncope

Y. J. KAO AND G. B. RACZ

Summary

A case of postanaesthesia micturition syncope with respiratory arrest is described. If syncope occurs, the temporary myocardial ischaemia and cerebral hypoperfusion may increase anaesthetic risk in the marginally compensated patient. The loss of airway protection during the syncopal period is also a cause of concern. We recommend the use of an indwelling bladder catheter during any prolonged surgical procedure.

Key words

Complications; bradycardia, hypotension.

Sudden decompression of an obstructed urinary bladder was associated with significant reductions in arterial blood pressure.¹ This change usually occurs over a 24- to 48-hour period, but it may occur immediately after decompression. The magnitude of the arterial pressure change is related to the initial degree of the distension.² A closely related clinical phenomenon, micturition syncope, was described in otherwise healthy patients.³⁻⁴ Typically, patients with this condition experience syncopal episodes associated with micturition. These episodes usually present as bradycardia, hypotension and loss of consciousness. The event usually lasts less than 30 minutes and recovery is always complete. Micturition syncope is a well established diagnosis in general medicine, but its occurrence after anaesthesia has not been described. A patient with syncope during the anaesthetic recovery period induced by micturition is reported.

Case history

A 48-year-old male presented for emergency laparotomy and possible appendectomy. Pre-anaesthetic examination revealed a well-developed, well-nourished male in mild distress. His temperature was 38.2°C, pulse 120 beats/minute, arterial blood pressure 155/85 mmHg and weight 68 kg. He arrived in the operating theatre with a nasotracheal sump tube in place and was receiving lactated Ringers' solution with 5% dextrose at a rate of 125 ml/hour. Laboratory tests revealed a haemoglobin level of 11.3 g/100 ml and a haematocrit of 32.4%. Monitoring included

ECG, automatic blood pressure, pulse oximeter and precordial stethoscope. A rapid sequence induction of anaesthesia was performed, after 3 minutes pre-oxygenation and Sellick's manoeuvre, with sufentanil 50 µg, hyoscine 0.2 mg, thiopentone 225 mg and suxamethonium 100 mg. Tracheal intubation with a 7.5-mm cuffed tube was uneventful. The patient's lungs were ventilated with N₂O 70% in oxygen. End-tidal CO₂ was maintained at 4.0 kPa by adjusting minute volume. The patient's vital signs remained stable throughout the induction period. Muscle relaxation was achieved with atracurium in 10 mg bolus (0.15 mg/kg) and monitored with a peripheral nerve stimulator. The patient also received sufentanil 25 µg every 30 minutes.

When the peritoneal cavity was opened, it was found to contain 2000 ml blood which was removed by aspiration. Additional 1500 ml of blood was removed over the next 30 minutes before haemostasis was achieved. It was discovered that the patient had a ruptured spleen and a laceration on the left lobe of the liver. Arterial blood pressure rapidly decreased to 90/60 mmHg. Another intravenous cannula was inserted while waiting for blood from the blood bank, and, over the next 30 minutes, 6 litres of warm lactated Ringers' solution were infused. The patient was also given 3 units of packed red blood cells after the blood arrived. Throughout the period of fluid resuscitation, blood pressure ranged between 125/75 and 90/60 mmHg, and finally stabilised at 125/75 mmHg.

Surgery was completed 150 minutes after the initial skin incision. Residual neuromuscular blockade was reversed

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This work was supported in part by the Roberts Foundation, Big Spring, Texas, USA.

Accepted 23 January 1990.

with edrophonium 40 mg preceded by glycopyrronium 0.4 mg. Nalbuphine 10 mg was given to reverse the potential respiratory depression effect of sufentanil (total dose 175 µg). The patient recovered from anaesthesia after the discontinuation of nitrous oxide, followed oral commands and his trachea was extubated. Upon awakening the patient's pulse was 90/minute, arterial pressure 140/85 mmHg, arterial O₂ saturation 98% and he complained of the urgent need to empty his bladder. The patient, after 30 seconds of continuous complaint, passed a large volume of urine (on the table) for 30 seconds to one minute. He lost consciousness immediately after voiding, developed bradycardia (heart rate 30/minute), his pulse became thready and respiration ceased. Electrocardiogram revealed sinus bradycardia but showed no evidence of conduction defects or ST segment changes. His lungs were immediately ventilated with 100% O₂. Arterial oxygen saturation increased from 70 to 98% and the trachea was re-intubated. Arterial blood pressure recovered to 120/76 mmHg with pulse of 75 beats/minute. However, he remained unconscious. Naloxone 0.2 mg was given intravenously but without immediate response. The patient developed sinus tachycardia to a rate of 140/minute over the next 10 minutes and hypertension 210/110 mmHg. Two doses of 15 mg labetalol were given and he stabilised with a pulse rate of 110 beats/minute and arterial pressure of 160/90 mmHg. He slowly regained consciousness, maintained stable vital signs and his trachea was again extubated. He was admitted to the surgical intensive care unit for observation. Chest X ray on admission to the intensive care unit showed no acute changes. All laboratory studies including serum electrolytes, cardiac enzymes, and blood gases were within normal limit, except haemoglobin level at 9.5 g/100 ml and haematocrit level of 29%. The rest of his recovery was uneventful.

Discussion

Loss of consciousness in the immediate postanaesthesia period may be caused by metabolic derangement, hypoxia, pharmacological effect or cardiovascular decompensation.⁵⁻⁷ Hypoglycaemia is the most frequently encountered cause of sudden alteration of consciousness, usually in medically treated diabetic patients who had a period of fasting but without intravenous glucose therapy.⁸ A sudden discontinuation of total parenteral nutrition can also precipitate hypoglycaemia. Hyponatraemia after transurethral resection of prostate can cause altered consciousness.⁹ None of these diagnoses apply in our patient.

Hypoxaemia can cause the loss of consciousness and haemodynamic decompensation.⁵⁻⁶ In our patient, arterial oxygen saturation was well maintained until the onset of cardiovascular decompensation and loss of consciousness. The subsequent arterial oxygen desaturation is probably the result of the syncope. This conclusion is supported by physical examination and chest X ray evaluation which failed to reveal any pulmonary pathology. No medication was given between emergence from anaesthesia and the onset of syncope. He had received a narcotic-nitrous oxide balanced anaesthesia. Sufentanil is a potent synthetic narcotic with a short half-life and so patients anaesthetised with it usually recover from respiratory depression earlier than patients anaesthetised with fentanyl. The failure of naloxone promptly to reverse the respiratory depression

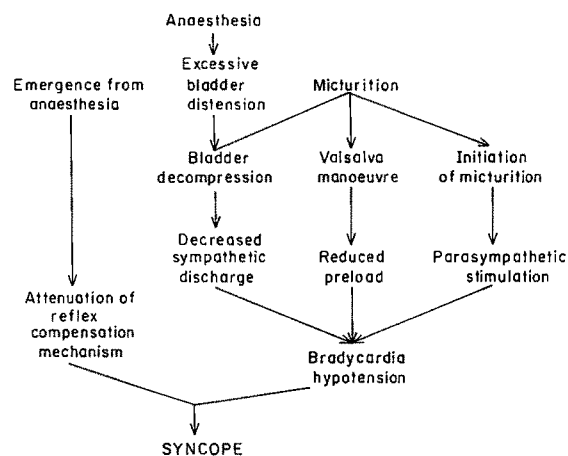


Fig. 1. Schematic summary of physiological changes that might have contributed to postanaesthetic micturition syncope.

and central nervous depression argued against a diagnosis of renarcotisation.

The most likely explanation for the syncopal episode is sudden cardiovascular decompensation. The patient was not experiencing acute volume shift or hypovolaemia at the time of the loss of consciousness, and had no evidence of myocardial ischaemia or infarction, nor any evidence of cardiac conduction disorder, so the possibility of reflex micturition syncope deserves to be considered. The observation of bradycardia and hypotension, the speed of the recovery and the temporal relation to micturition are all consistent with the diagnosis of micturition syncope.¹⁰⁻¹¹

Proudfit and Forteza were the first authors to call attention to micturition syncope.³ The classical presentation of micturition syncope is to air force physicians. They describe relatively healthy young males who, after several hours of recumbency, arose to pass urine and suffered syncope.² No cardiovascular and neurological abnormalities were found. Recently, it became clear that micturition syncope may also occur in female and elderly patients. Syncope with bradycardia and hypotension after micturition remains the only common presentation.^{10-11,14}

We believe the mechanisms (Fig. 1) were responsible for the observed syncope. A distended bladder is a strong, afferent stimulus to the central nerve system. Sudden reduction of the peripheral vascular tone may result from the diminution of sympathetic tone.¹² Therefore, it is unsurprising that a reduction of blood pressure follows emptying the urinary bladder.¹⁻² Voiding may be initiated with Valsalva manoeuvre producing an increase in intrathoracic and intra-abdominal pressure and, consequently, reducing venous return which in some patients produces hypotension and syncope.³ Parasympathetic activation via vagal discharge produced by the act of micturition can further decrease the already low blood pressure.¹³ Residual anaesthetics may attenuate the physiological homeostatic reflexes.

Acknowledgment

The authors wish to thank Mrs C. Barnes for her secretarial assistance in the preparation of the manuscript.

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CASE REPORT

Withdrawal syndrome after propofol infusion

J. AU, W. S. WALKER AND D. H. T. SCOTT

Summary

We report a case of classical general depressant withdrawal syndrome that culminated in grand mal convulsions, in a patient who received propofol infusion for sedation after cardiac surgery.

Key words

Complications; convulsions.

Anaesthetics, intravenous; propofol.

Propofol/(Diprivan) has been widely investigated for use in the induction and maintenance of general anaesthesia.¹ More recently it was used as an intravenous sedative during gastrointestinal endoscopies,² spinal anaesthesia,³ and in the intensive care unit (ITU),^{4,5} with minimal adverse effects. We report a patient who developed a classical general depressant withdrawal syndrome⁶ that culminated in several grand mal convulsions, after the use of propofol for sedation in the intensive care unit.

Case history

A 41-year-old man presented to hospital with a history of sudden onset of central chest pain associated with left lower limb monoplegia. There was no past medical history of note, and in particular no history of alcohol abuse or epilepsy. He had a weak left radial and an absent left femoral pulse, and aortic systolic and diastolic murmurs. Chest X ray demonstrated a widened mediastinum. Aortic dissection was diagnosed and confirmed by aortography, which showed free aortic reflux and an extensive dissection that extended from ascending to abdominal aorta. He was taken to theatre for emergency surgery.

Anaesthesia was induced with thiopentone and maintained with nitrous oxide in oxygen, and morphine. Neuromuscular block was started with atracurium and maintained with boluses of pancuronium. Resuspension of the aortic valve and replacement of the ascending aorta and aortic arch were performed under cardiopulmonary bypass, deep hypothermia (16°C), and circulatory arrest. Cerebral protection during 60 minutes of circulatory arrest was effected by thiopentone 3 g, 20% mannitol 100 ml, and hydrocortisone 1 g.

Artificial ventilation of the lungs was continued electively after operation and he remained haemodynamically stable. Spontaneous eye opening occurred within 12 hours and he responded to simple commands. Sedation was, however, required for increasing agitation, and infusions of papaveretum and propofol were commenced. He was successfully weaned from the ventilator and extubated after 5 days. Sedation was discontinued. He became increasingly confused, tremulous, and suffered hallucinations over the next few hours. Ten hours later he developed several short-lived grand mal convulsions that responded to an intravenous injection of diazepam. Papaveretum and propofol infusions were recommenced, and gradually withdrawn over the next 48 hours without the recurrence of fits. An EEG at this stage demonstrated no evidence of epileptic activity. However, he remained in an organic delusional state for another 7 days before regaining full mental function. He was eventually discharged home 23 days after operation.

Discussion

Propofol was reported to induce epileptiform activity in known epileptics when used as an anaesthetic agent,⁷ and convulsions were also reported after its use in such patients.^{8–10} A case of convulsion was also reported after propofol anaesthesia in a patient with no history of epilepsy.¹¹ Other workers, on the other hand, have reported its use in the treatment of status epilepticus,¹² and its effect on shortening seizure duration when used for electroconvulsive therapy.¹³ The role of propofol in epileptogenesis is therefore controversial.

Convulsions apparently occurred in our patient as a

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Accepted 22 February 1990.

result of sedative withdrawal. Both papaveretum and propofol infusions were used, but the clinical features upon withdrawal were classical of the general depressant withdrawal syndrome,⁶ and incompatible with opioid withdrawal. Convulsions can also complicate cardiopulmonary bypass with deep hypothermia and circulatory arrest, but typically these seizures are transient and followed by uneventful convalescence.¹⁴

There have been two previous reports of the use of propofol infusion for short-term sedation in the ITU, with rapid recovery and minimal adverse effects upon drug withdrawal.^{4,5} The dosages used were similar to those in our patient, although the duration of infusion was much less (8 hours as compared with 5 days). Our patient demonstrates that the prolonged use of propofol infusion for sedation may be associated with the rapid development of drug dependence, and the occurrence of the general depressant withdrawal syndrome upon discontinuation of therapy. We recommend that therapy should be withdrawn cautiously in such patients, and that signs of withdrawal be sought actively.

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CASE REPORT

Prolonged neuromuscular blockade with vecuronium in a neonate with renal failure

S. R. HAYNES AND N. S. MORTON

Summary

An 11-day-old neonate with renal failure caused by dysplastic kidneys was anaesthetised with thiopentone, vecuronium, nitrous oxide and oxygen, for insertion of a long-term peritoneal dialysis catheter. Complete neuromuscular block of 210 minutes' duration ensued after the initial dose of vecuronium (97 µg/kg). Partial block persisted for a further 30 minutes. The prolonged neuromuscular block in this case may have been because of proportionately greater dependence on renal clearance of vecuronium in neonates.

Key words

Kidney; failure.

Neuromuscular relaxants; prolonged action.

Vecuronium is widely used to produce muscle relaxation in patients with renal failure. It is also extensively used to produce muscle relaxation in neonates who have surgery, and was found suitable for use in both groups of patients. This report describes the use of vecuronium in a term infant with established renal failure.

Case history

An 11-day-old male presented for insertion of a long-term peritoneal dialysis catheter under general anaesthesia. He was delivered at term, weighing 3.2 kg, by spontaneous vertex delivery. Subsequently he had become lethargic, and was oliguric with elevated plasma urea and creatinine levels. Dysplastic kidneys were identified by ultrasound, but their appearance was not that of infantile polycystic disease. Initially, he was managed medically, but it was believed that he would require long-term peritoneal dialysis.

On the day of surgery he weighed 3.15 kg, with plasma Na⁺ 147 mmol/litre; K⁺ 4.4 mmol/litre; urea 12.6 mmol/litre; creatinine 1225 mmol/litre, and Ca²⁺ 2.48 mmol/litre. Capillary blood gas analysis showed pH 7.31, P_{O₂} 6.2 kPa, P_{CO₂} 8.6 kPa, and base excess -2.0 mmol/litre. He was very mildly jaundiced, and the plasma bilirubin was 117 mmol/litre. This was not considered to represent anything

other than 'physiological' neonatal jaundice. The patient was not receiving any medication known to prolong the duration of action of non-depolarising muscle relaxants.

Anaesthesia was induced with thiopentone 12 mg, vecuronium 300 µg, nitrous oxide and oxygen. The trachea was intubated and the lungs hand ventilated with a Mapleson F system. It was anticipated that the procedure would require an anaesthetic lasting 40–60 minutes. The patient was monitored with a precordial stethoscope, ECG, pulse oximeter, an automatic blood pressure cuff (Dinamap), rectal temperature probe, and a transcutaneous nerve stimulator. Electrodes were placed over the ulnar nerve and the adductor pollicis twitch observed clinically in response to a supramaximal train of four stimulus at 2 Hz. An overhead radiant heater was in position during induction and after operation, and the baby was placed on a heated water blanket. Surgery was completed 40 minutes after induction. Normothermia and cardiovascular stability were maintained throughout. No further increments of vecuronium were given.

There was no evidence of reversal of neuromuscular block on completion of surgery when neostigmine 150 µg and atropine 75 µg were given. The patient was kept in the operating theatre, ventilated, for a further 40 minutes, after which neostigmine 75 µg and atropine 40 µg were given, again with no evidence of reversal. The patient was

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Accepted 6 February 1990.

returned to the ward ventilated. The first sign of reversal of neuromuscular block was seen at 210 minutes after induction of anaesthesia, when there was a single twitch response to a train-of-four stimulus, which indicated a 90% neuromuscular block. Recovery continued, and 240 minutes after induction there was no residual neuromuscular blockade. The baby was, however, intermittently apnoeic and required ventilation overnight. The next day the plasma K^+ was 6.6 mmol/litre and creatinine 1217 mmol/litre and it was believed necessary to keep the child intubated until there was some biochemical improvement. The tracheal tube was eventually removed 36 hours after the induction of anaesthesia.

Subsequently the baby has remained jaundiced; the plasma bilirubin has stayed at 100–130 μ mol/litre, of which approximately 50% is conjugated. The highest plasma AST estimation was 79 IU/litre, and the highest ALT 32 IU/litre; both results were within the hospital's normal neonatal range. The coagulation screen has always been normal. The baby's stools were normally pigmented, and ultrasound examination of the biliary tree has shown a functioning gall bladder with no evidence of any anatomical abnormality. No cause was identified for the prolonged jaundice despite extensive investigation. A liver biopsy, however, has not been performed. The patient remains alive at the time of writing and is receiving regular peritoneal dialysis.

Discussion

Neonates exhibit a wide inter-individual variation in response to non-depolarising relaxants; some are sensitive to very small doses, and others are relatively resistant. It is generally held that sensitivity is related to immature function of the neuromuscular junction. It has been demonstrated that the plasma concentration of tubocurarine that causes 50% depression of the twitch response is markedly lower in neonates than in adults.¹ No such data are available for vecuronium. However, since neonates have proportionately twice the extracellular fluid volume of adults, the volume of distribution of water-soluble drugs will be greater. The degree of protein binding of some muscle relaxants is less in neonates, and results in greater efficacy at lower plasma concentrations.²

It was shown recently that vecuronium in a dose of 100 μ g/kg in infants under 3 months of age causes a 90% blockade for a mean duration of 59 minutes.³ However, this study did not include neonates as a separate group.

The neostigmine requirement of infants and children is less than half that of adults.⁴ However, this observation was made when the twitch height had spontaneously returned to 10% of normal; no comment was made about the efficacy of neostigmine where there is no spontaneous return of function.

Vecuronium is widely used in patients with renal failure. Pharmacokinetic and pharmacodynamic studies of vecuronium in patients who have renal transplantation demonstrated increased distribution and elimination half-lives, and decreased clearance.⁵ Three of the patients with renal failure studied by this group had complete neuromuscular block 150 minutes after a dose of 100 μ g/kg had been given. All three patients were easily reversed by neostigmine and stopping the administration of isoflurane.

The volume of distribution of vecuronium is greater than the extracellular fluid volume, so there is evidently exten-

sive tissue localisation, probably in the liver. Biliary vecuronium concentrations have been shown to be high 20 minutes after administration.⁶

Renal failure can affect the pharmacokinetics of drugs in various ways: these include decreased clearance, low plasma albumin due to increased protein losses, altered protein molecular structure, and the effect of other endogenous substances, which when present in greater concentrations in renal failure compete for drug-binding sites. Most of the above discussion is related to adult practice, but there are fewer data available on paediatric usage of vecuronium. It was anticipated that neuromuscular block would last longer than usual in a child of this age, but it was not expected to be as prolonged nor to be as resistant to neostigmine as it was in this case.

Data derived from adult practice indicate that 80–90% of vecuronium is metabolised by the liver and that only 10–20% is cleared by the kidneys.⁷ This case suggests that this may not be applicable in neonates. There may be a proportionately greater dependence on renal excretion in this age group. It was not considered that any hepatic pathology was present when this patient was anaesthetised. The combination of hepatic and renal failure would have explained the prolonged neuromuscular block, if there was a significant degree of hepatic dysfunction at the time of anaesthesia, and vecuronium would have been contraindicated. This, however, would not account for the resistance of neostigmine.

No cause was identified for this patient's jaundice despite extensive investigations, and there is no evidence of overt hepatic failure. We believe that it is unlikely that hepatic dysfunction had a significant role in the genesis of the prolonged neuromuscular block described. Prolonged neuromuscular block has been reported after infusion in an adult with renal failure,⁸ and also after infusion in a 2-month-old infant with normal renal function.⁹

An alternative anaesthetic technique could have been used in this case. It could be argued that the dose of vecuronium administered was excessive and a smaller dose would have been more appropriate. An alternative muscle relaxant could have been used; the obvious choice is atracurium. Atracurium degrades spontaneously by Hoffman elimination, but it is associated with the potential problems of histamine release and laudanosine production. Conscious intubation or intubation under halothane anaesthesia would have avoided the need for a muscle relaxant.

We conclude that vecuronium in the neonate with renal failure may result in very prolonged neuromuscular block.

Acknowledgment

We thank C. Roulston for her assistance in the preparation of this manuscript.

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The Engström Elsa anaesthetic machine

An electronic system for anaesthesia

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Summary

Six Engström Elsa anaesthetic machines have been in regular use for 18–24 months. The machine incorporates a number of new concepts for anaesthetic delivery and monitoring. At flows below 1000 ml/minute, each machine delivered 20% more than the indicated value; at higher flows, the indicated value was within 10% of the flow delivered. Minute volume, tidal volumes and oxygen concentrations were within the manufacturer's specifications. However, vaporizer and vapour monitor performance was outside the (SD) 5% accuracy claimed by the manufacturers. It was noted that the bistable valve requires user familiarity for the change from controlled to spontaneous ventilation to be accomplished with ease. It was also possible to misconnect the breathing system and so isolate the excess pressure escape valve and high-pressure alarm. Nevertheless, once familiarisation was achieved, the machines have proved easy to operate and are particularly satisfactory when used with low fresh gas flows.

Key words

Equipment; machines.

There has been considerable interest in recent years in the design of anaesthetic machines which incorporate means of economy in the use of volatile and gaseous anaesthetic agents and reduction of environmental hazards. The use of electronic (microprocessor) systems to provide accurate monitoring and display of the major variables of interest has also been deemed appropriate.¹ Engström (Gambro Engström AB, Sweden) with these goals in mind, have introduced a new anaesthetic machine, the Elsa (acronym for Electronic System for Anaesthesia), which combines a number of novel features. The machine is considered to be suitable for low flow or semiclosed anaesthesia with carbon dioxide absorption, or alternatively conventional semiclosed systems of the Mapleson A or D types, and applicable to both adult and paediatric practice. The machine has independent parallel systems to eliminate the consequences of any single fault.

Overall design

There is a high degree of technical integration; in addition to the major functions such as the ventilator, vaporizer and fresh gas generator, the sensors for volume, pressure and gas concentration measurements in the patient system are all built into the machine. There are few external con-

nexions. The Elsa can be considered in two parts: the delivery unit and the monitor unit. There is a large shelf for additional patient monitoring equipment above the monitor unit. The top of the delivery unit provides a generous working surface and a large drawer at the bottom of the machine provides adequate storage space.

On each side are two monitoring rails; the lower one is of the 'swing out' type, intended for mounting carbon dioxide absorber and suction units. The upper rails are continuous with handles on each side which allow easy movement of the machine (Fig. 1). The reservoir bag, enclosed in a transparent 'bottle', is in view on the left hand side, as is the manual bag and all patient-related outlets (Fig. 2). Gas supplies (cylinder and pipeline) are connected at the rear and three different liquid volatile agents in their original bottles are mounted above the cylinders. The delivery unit is divided into three functional fields: fresh gas flow control, vaporization and ventilation. There are knobs for setting alarm levels across the top of the panel under a hinged cover. The main ventilation and gas concentration variables are shown in the lower half of the monitor unit panel while the upper field of the panel displays a 24-hour clock and display used for alphanumeric information, in particular alarm messages. Each functional block will be considered in detail.

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Accepted 23 January 1990.

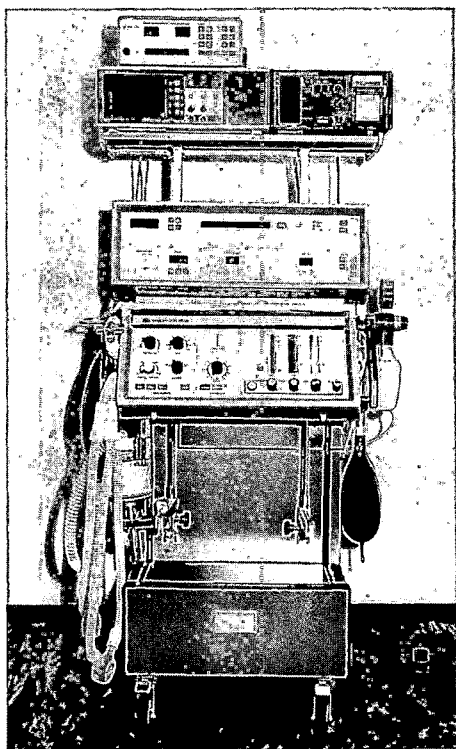


Fig. 1. The Engstrom Elsa. Patient monitors occupy the instrument shelf.

Delivery unit

Gas supply

Three different gases can be delivered, oxygen (O_2), nitrous oxide (N_2O) and one other, which can be air, nitrogen or carbon dioxide. Normally gas is obtained by pipeline (250–500 kPa). Up to four cylinders can be mounted on the back of the machine. Whatever the source, built-in regulators stabilise the working pressure to 200 kPa. An N_2O/O_2 slave regulator shuts off the N_2O supply should the O_2 pressure decrease. Nitrous oxide and driving gas pressures are monitored electromechanically, and a fall in O_2 pressure triggers a pneumatic alarm whistle. Either air or O_2 can be used to drive the ventilator and vaporizer, while O_2 is used as the driving gas in the manual bag.

Flow control system

Each gas flow is controlled by a mechanical needle valve. The set value is measured by a thermistor flowmeter and the result displayed by a vacuum fluorescent display as an appropriately coloured bargraph. Accurate settings are claimed at flows down to 200 ml/minute. The mixer provides a shunt flow of 200 ml/minute O_2 , which provides an internal calibration system for checking the sensor in the O_2 flowmeter. There is an O_2 flush facility. Total fresh gas flow is measured by a separate thermistor flow-meter. An electronic shut-off valve stops all gas flow when standby conditions are required, and under standby conditions another pressure-operated valve can be opened to allow a flow of 100 ml/minute O_2 to test the circle system for leaks.

Vaporizer

Normally, three volatile anaesthetic agents, halothane, enflurane and isoflurane are connected in their original

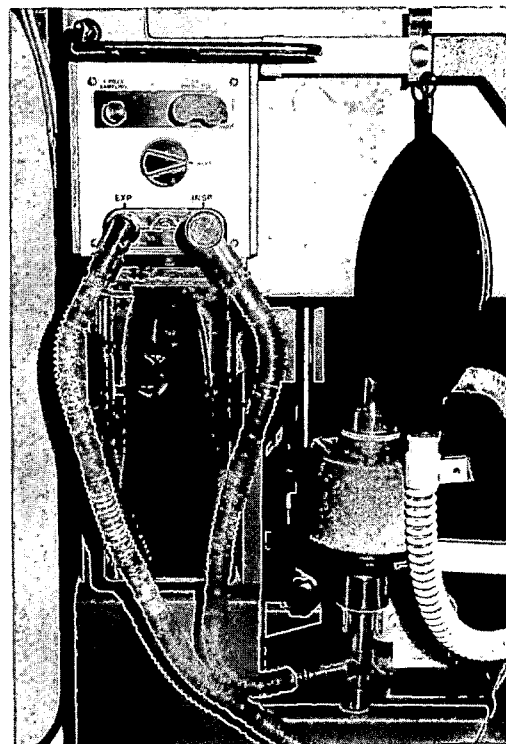


Fig. 2. Left sideview of Elsa showing the patient unit panel, the bag-in-bottle assembly, and the manual bag. The fresh gas outlet selector, which conceals the external fresh gas outlet, is at the top right of the patient unit panel.

bottles. A special cap for each is fitted with a capacitive electronic sensor which enables the liquid level in the bottle to be displayed on the front panel of the delivery unit. Only one agent can be administered at any one time. Driving gas at a fixed pressure forces pulses of liquid anaesthetic into a heated vaporizing chamber (with an audible click) at a rate depending on the demand for the agent, from which anaesthetic vapour is carried into the patient breathing system. The driving gas is automatically cut off if abnormally high anaesthetic concentrations are detected or malfunction of the vaporizing chamber occurs. The concentration of volatile agent is sensed by a specially coated crystal which is part of an oscillator system; frequency of oscillation changes according to the concentration of anaesthetic agent present at the crystal. A Peltier element and thermistor maintain the temperature of the complete assembly at approximately 70°C. Volatile agent concentrations can be monitored at one of three points in the breathing system; calibration is automatic using a specially supplied test can (Elsacan) which contains 1.5% halothane and zeroing is also automatic.

Ventilator and circle breathing system

The ventilator block works on the bag-in-bottle principle. The mechanical ventilation and the manual ventilation/spontaneous breathing systems are connected to the transparent pressure chamber via a two-way bistable mechanical valve which switches from one position to the other according to the pressure difference between its inputs. A valve allows adjustment of gas volume in the manual bag. A time-cycled valve in conjunction with a differential pneumotachograph controls the rate and volume of gas delivered to the patient during mechanical ventilation, and a

spring-loaded valve allows venting of excess breathing gas during the expiratory phase. There is facility for using positive end-expiratory pressure (PEEP) and extended mandatory minute volume (EMMV). The reservoir bag assembly is autoclavable, and expired gases pass through a scavenging port.

Fresh gas supply is normally routed internally to the circle breathing system. A fresh gas outlet on the patient panel allows suitable external breathing systems to be connected; a Bain system appropriate to Elsa is available, which allows the machine's own ventilator to operate if required. A lever is opened to allow external systems to be connected, and thus switches the fresh gas flow to the outlet (Fig. 2).

Fresh gas flow, tidal volume and minute volume are measured by separate flow-sensing thermistors each of which forms one arm of a bridge system. Reference thermistors measure the temperature of the gas passing the flow sensors and appropriate microprocessor compensation is made for changes in apparent flow rates which may be the result of temperature fluctuations. Oxygen concentration monitoring uses a paramagnetic oxygen analyser employing an optical sensing device. Linearity of scale allows calibration by two point checks only. Oxygen concentration can be monitored at any one of three points in the breathing system, as with the volatile agent. Airway pressure is measured by a pneumatic analogue meter.

Monitoring safety and alarm features

The machine, when it is first switched on, can be programmed to run through a pre-use checklist which is displayed in abbreviated form on the alphanumeric display. This routine is optional and may be dispensed with. The machine then automatically checks its memory routines and electronic hardware while a pre-use check message is displayed. It switches to stand by after about 30 seconds and is then ready for use by selection of the required mode by pressing the appropriate soft key. The manufacturers recommend that a pre-use tightness check is performed before each anaesthetic to test the integrity of the circle system.

The machine monitors airway pressure, fresh gas flow, minute and tidal volumes, and the concentration of volatile agent and O₂ during use. High and low alarm settings can be chosen for each of these measurements, and initiation of an audible alarm is accompanied by a visual message to indicate the problem. Failure of N₂O and driving gas also actuates alarm messages and an acoustic alarm. Failure of the O₂ supply causes a pneumatic alarm to whistle and automatically shuts off the N₂O supply. A delivered hypoxic fresh gas mixture will also start an alarm and if corrective action is not taken within 30 seconds, the N₂O flow is shut off. In the event of electrical power supply failure, the electronic bargraph, which indicates gas flow will, of course, disappear, but O₂, N₂O and air can still be delivered and adjusted using a rough scale on the rotameter knobs, and manual ventilation can be performed.

Other features

A change from controlled to manual ventilation requires an increase in airway pressure to change the position of the bistable mechanical valve. This is effected by squeezing the

manual bag, when an audible click is heard, and patient breathing will be seen reflected in movements of the manual bag. All parts of the breathing system that come in contact with expired gases can be removed and either autoclaved, gas sterilised, or cleansed in disinfectant. The complexity of the machine will be appreciated when it is realised that there are over 700 internal system interconnexions. An RS232 interface allows automatic printout of data by a stand-alone printer or connexion to a computer for data collection. There is a separate connexion for an analogue data recorder.

Results

Six Elsas were installed in a new multitheatre suite over a period of 6 months and have been in constant use for 18–24 months. Approximately 15 000 adult patients have been anaesthetised. Major malfunctions which required temporary removal from service occurred approximately once in 18 months for each machine. Minor problems were easily rectified by our medical physics technicians once they gained familiarity with the machines. The anaesthetists, once they became familiar with the machines, also found them extremely easy to use. However, like most of the new generation of electronic machines that are currently being marketed, a period of practical familiarisation is required for all staff who have no previous experience of the machine. It is not a machine to be left in theatre for the on-call anaesthetist to use for the first time for an emergency ruptured aortic aneurysm!

The design of the vaporizer unit is different from the conventional plenum type, and build up of concentration takes several minutes if the machine is started from 'cold'. This problem can be overcome by turning on a low gas-flow concentration of anaesthetic vapour while the pre-induction preparations are made or the patient is being induced in the anaesthetic room. The knob that increases the concentration of the volatile agent in our machines has to be moved in a clockwise direction, which is against common convention for mechanical control of vapours and gases, but is the electrical convention; this has been changed in later models.

We do not restrict the use of isoflurane in our hospital, so the drug is widely used. There was a major price rise in the cost of the agent during the period when the Elsas were being installed; in spite of this, the amount of isoflurane used decreased to 40% of previous amounts and there was a slight overall monetary saving. Most of our clinicians favour total gas flows of 2–4 litres/minute but our personal experience convinces us that the use of total fresh gas flows of less than 1 litre/minute is satisfactory and that the machine's internal monitoring ensures the safety of low flow techniques. A sensitivity $\times 10$ key allows a 10 times magnification of the bar graph display when low flows are set. Use of low flows might justify the manufacturer's claim that the purchase price of the machine could be reclaimed by savings in the cost of expensive volatile anaesthetics in 2–3 years, when compared with the cost of semiclosed or nonbreathing techniques.

Our machines did not undergo routine servicing during the period covered by this review because of delays in delivery of specialised equipment. Each machine was tested on at least two occasions after 18–24 months regular daily use to check the accuracy of fresh gas flows, inspiratory

tidal and expiratory minute volumes, vaporizer output, vapour concentration monitors, and oxygen concentration monitors.

Some problems have been noted during use and in discussion with colleagues. When returning from the CMV mode to spontaneous respiration, or at the beginning of an anaesthetic employing spontaneous respiration, the manual bag requires a gentle squeeze to move the bistable valve to the open position to allow respiratory movements to be seen in movements of the manual bag.

Attempts have been made to ventilate the patient's lungs when the external fresh gas selector and outlet is held open by an attached semiclosed system or O₂ lead. No fresh gas reaches the ventilator system and the machine alarms with a patient 'bag empty' signal. Additionally, the recommended tightness check can be performed satisfactorily with the external fresh gas outlet held open with no indication that fresh gas is bypassing the circle system. We believe that the manufacturers should introduce a warning signal to indicate that the gas port is open.

Individuals unfamiliar with the machine have experienced difficulty in manually ventilating patients during induction of anaesthesia, when the manual bag is over-inflated, thus forcing all gas out of the reservoir bag. This situation may have arisen because of the somewhat concealed position of the reservoir bag/bottle assembly and would be less likely to occur with an upright bellows ventilator.

We are aware of a critical incident that arose in another hospital when an unskilled person attached the expiratory limb of the patient Y-piece to the external fresh gas outlet. This manoeuvre isolates the high-pressure alarm and the excess pressure escape valve. The patient survived with bilateral pneumothoraces. The manufacturers are aware of the fact that this problem has arisen, and are considering whether modifications would be appropriate. This error would be easily recognised if the tightness check were performed routinely before each anaesthetic.

In general, however, our overall impression of these machines after considerable experience is favourable. They have proved easy to operate and user friendly once familiarisation is achieved. Reduction of external gas connexions to the minimum means that the opportunities for accidental disconnexions are considerably reduced, and the

internal monitoring facilities seem to be reliable and should be an important contribution to patient safety. Our own experiences with low flow techniques support the claim that the machines are capable of making a substantial contribution to economy in the use of anaesthetic gases and vapours.

Performance assurance and validation (See Table 1)

Each experiment was repeated on each machine on at least two separate occasions.

Fresh gas flows were measured at the external gas outlet using an RT-200 Calibration Analyzer (Timeter Instrument Corp, Oregon, Pennsylvania). Equal parts of N₂O/O₂ were used, at low flows which started at displayed gas flows of 400 ml/minute and increased in steps of 200 ml to 1000 ml/minute, and at higher flows which started at 1.5 litres/minute and increased in 0.5-litre steps to 10 litres/minute. The RT-200, which was a new instrument under guarantee has a stated accuracy of (SD) 5% at low flows, and (SD) 4% at high flows with these gas mixtures. Table 1 shows that at low flows (400–1000 ml/minute) each machine delivered on average 20% more gas than the gas display indicated, while at higher flows all machines were within the manufacturer's claimed tolerances. Most accurate flows were obtained within the range 1.5–3 litres/minute.

Inspired tidal volumes and expired minute volumes were measured with a Magtrak Respiratory Monitor (Ferraris Development and Engineering Co Ltd). This instrument had been previously compared with the RT-200 (which is not suited to making measurements within a ventilator circle) using a minute volume divider ventilator and found to over-read on average 10%. The Magtrak electronic flow sensor was appropriately positioned, depending on whether inspiratory or expiratory volumes were being measured, next to a dummy lung attached to the ventilator Y-piece, and the ventilator set to deliver in turn 400, 600 and 800 ml tidal volume at 10 breaths/minute with a fresh gas flow of 4 litres/minute containing 50% N₂O in O₂. The mean of at least three readings was taken. The machine reading of tidal and minute volumes agreed closely with the ventilator settings and were within the (SD) 7% accuracy claimed. Figure 1 gives the differences between the Magtrak monitor and the machine reading at the 600-ml tidal volume setting.

Table 1. Summary of measurements made to validate the performance of six Elsa anaesthetic machines after 18–24 months regular use without routine service.

Variable	Average difference from test equipment (%)	Range of differences (%) (six machines)	Manufacturer's claimed accuracy (%)
Fresh gas flow N ₂ O/O ₂ 400–1000 ml/minute	–19	–11 to –28	±10
Fresh gas flow N ₂ O/O ₂ 1.5–10 litres/minute	±7	+8 to –10	±10
Tidal volume	+5	+2 to +9	±7
Expired minute volume	+6	0 to +11	±7
Vaporizer at 2% settings			
Halothane	±8	+10 to –10	±5
Enflurane	±25	+10 to –55	±5
Isoflurane	±9	+10 to –20	±5
Vapor monitor			
Halothane	+12	0 to +22	±5
Enflurane	+18	+6 to +32	±5
Isoflurane	+17	+6 to +33	±5
Oxygen monitor	±1.3	+4 to –2	±2

Results at the 400 and 800 ml settings were broadly similar. Vaporizer outputs were measured and anaesthetic agent monitors and O₂ concentration measurements checked using a Siemens Servo 120 Gas Monitor (Siemens-Elma AB, Sweden). Readings of the latter agreed within 3% with the outputs of two recently serviced halothane and isoflurane vaporizers at the 1%, 2% and 3% settings.

Measurements were made at the external fresh gas outlet of the Elsa with a gas flow of 4 litres/minute of 50% N₂O in O₂ and the vaporizer of the Elsa set at 1%, 2% and 3% in turn for halothane, enflurane and isoflurane. Measurements were taken when steady state conditions were established, with the Servo 120 switched to the appropriate agent. Oxygen concentrations, as measured with the Servo 120 (the O₂ cell having been calibrated with air), were compared with the machine reading of O₂ concentration in the fresh gas flow.

Table 1 lists the findings at the 2% setting for each agent. Measurements made at the 1% and 3% settings showed variations in vapour output and vapour monitor readings which were almost identical for each machine. The vaporizer output for halothane for all six machines did not

exceed (SD) 10% of set output; the situation with isoflurane was almost as good, with only one machine delivering 20% less than the set 2%. Three machines delivered considerably less than the set concentrations of enflurane. There were rather wide variations in the vapour monitors and no single machine approached the manufacturer's claimed accuracy of (SD) 5% of read value for all three anaesthetic vapours. Measurements of O₂ concentration were in substantial agreement (Table 1).

Acknowledgments

We are grateful to our anaesthetist colleagues of all grades who discussed their experiences with these machines and gave us the benefit of their opinions.

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Resistance to fluid flow through two spinal needles

P. J. TOOMEY

Summary

A new needle, 120 mm long and 26 gauge in diameter, has been introduced for subarachnoid anaesthesia. The resistance to flow through the needle was measured using a pressure monitor infusion pump and compared with the resistance to flow through a 90-mm 26-gauge needle; there was a threefold increase in resistance in the 120-mm needle compared to the 90-mm needle, which could not be explained by the difference in length alone and must reflect a decrease in the internal diameter of the long needle. This results in difficulty in identifying the subarachnoid space by backflow of cerebrospinal fluid.

Key words

*Anaesthetic techniques, regional; spinal.
Equipment; needles.*

A new, 120-mm long, 26-gauge (0.7 mm) spinal needle (Spinocan), manufactured by B. Braun Medical Ltd, has been introduced for spinal anaesthesia. It is intended to be inserted through an epidural Tuohy needle to facilitate combined subarachnoid anaesthesia with placement of an epidural catheter. However, we have noticed some technical difficulties during evaluation of this needle in obstetric practice. The time taken for cerebrospinal fluid (CSF) to appear in the hub of the needle was considerably longer in the 120-mm Braun needle than in the 90-mm 26-gauge Becton Dickinson needle (BD) we had previously used for spinal anaesthesia. It was noted in 10 consecutive cases of spinal anaesthesia performed by the same anaesthetist with the mother in the left lateral position, that the time between withdrawal of the stylette to the formation of a meniscus of CSF at the hub of the Spinocan had a mean of 141 seconds, with a range of 89–233 seconds, and 95% confidence intervals (CI) of 131 to 151 seconds.

The aim of this study was to determine the flow characteristics of the new 120-mm 26-gauge Braun needle and compare it to the data on the 90-mm 26-gauge Becton Dickinson (BD) needle as described by Morris and his colleagues.¹ They investigated the problem of differing flows through spinal needles by determining the resistance to flow of the needles using a pressure monitor infusion pump.

Methods

The 120-mm 26-gauge Braun Spinocan was compared with the 90-mm 26-gauge BD spinal needle in two sets of

experiments. The rate of fluid flow was determined using a constant head of pressure of 10 cm water. The head of pressure was produced by the barrel of a 50-ml syringe with its plunger removed and filled with compound sodium lactate solution. A three-way tap was connected to the tip of the syringe. Manometer tubing leading to a centimetre scale was connected to one port of the tap and a rubber stopper fixed to the other port (Fig. 1). The system was mounted in a horizontal plane on a bench top and the reservoir filled to a height of 10-cm as shown by the manometer scale.

The spinal needles were inserted through the rubber stopper, the stylette was removed and the times when the first and second drops fell from the hub of the needle were recorded with a stopclock. Five unused needles of each type were used.

The resistance to flow was determined using a pressure monitor infusion pump (IVAC 560). This is a variable pressure volumetric pump which delivers a preset volume of fluid at a set rate, with a pressure transducer incorporated into the giving set to produce a digital readout of the pressure within the system. Compound sodium lactate solution was used, the transducer was calibrated against a mercury column and the system zeroed to atmospheric pressure. The spinal needles under test were connected by the hub to the end of the giving set and held in a horizontal position. Two sets of pressure recordings were made at steady state conditions with the pump set to flow at 50 ml/hour and at 100 ml/hour. Five needles of each type were used. Resistance to flow was calculated for each needle: (resistance = pressure/flow).

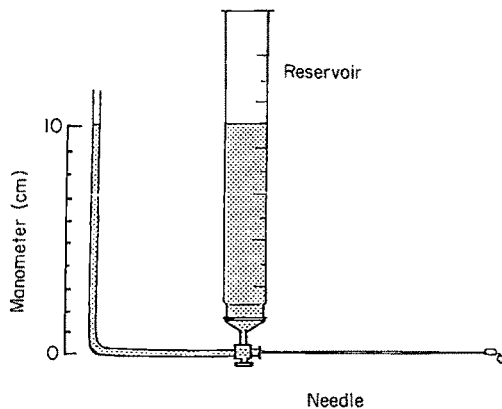


Fig. 1. Reservoir system used to measure the flow through needles.

Results

At a constant head of pressure of 10 cm water (Table 1), the times to the first drop from the 26-gauge BD needles had a mean of 94 seconds (CI, 89.9–98.1), with a mean interval between drops of 27 seconds (CI, 22.9–31.0). The Braun 26-gauge Spinocan had a mean time to first drop of 310 seconds (CI, 288–331), with a mean interval between drops of 77 seconds (CI, 69.7–84.3).

It was found that the mean resistance to flow (Tables 2 and 3) for the Spinocan 26-gauge 120-mm needle was 0.4 kPa/(ml/hour) (CI, 0.43–0.44). This was more than three times greater than the mean resistance of the BD 26-gauge 90-mm needle; 0.14 kPa/(ml/hour) (CI, 0.13–0.14).

Discussion

Various studies have been undertaken to measure needle flow-characteristics and flow of CSF and artificial CSF through spinal needles.

Gerrish and Peacock² studied 14 types of spinal needle from six different manufacturers and measured flow of an artificial CSF through the needles at a pressure head of 12 and 50 cm CSF to simulate the lying and sitting positions. They found that there were considerable dissimilarities in the needles and attributed variations in flow rate to the different internal diameters and the quality of finish of the internal surface, a feature demonstrated by Messahel *et al.*³ Morris *et al.* demonstrated that there was a significant difference in flow rates through a standard length 26-gauge Spinocan and a 26-gauge BD needle.¹ Palleiko *et al.*⁴ repeated the work of Morris *et al.* using a redesigned 26-gauge Spinocan and found flow rates and resistance to be indistinguishable from the BD 26-gauge needle.

Table 1. Time (seconds) to drops of fluid from hub at a pressure of 10 cm water.

	Braun needle		BD needle	
	1st drop	2nd drop	1st drop	2nd drop
Needle number				
1	306	86	92	32
2	299	81	95	23
3	306	70	89	26
4	340	72	99	30
5	298	75	93	25

Table 2. Pressure (kPa) and resistance to flow (kPa/(ml/hour)) at 50 ml/hour flow rate.

	Braun needle		BD needle	
	Pressure	Resistance	Pressure	Resistance
Needle number				
1	21.8	0.44	6.8	0.14
2	21.1	0.42	7.2	0.14
3	22.1	0.44	6.8	0.14
4	21.7	0.43	6.8	0.14
5	21.9	0.44	7.0	0.14

Cruickshank and Hopkinson⁵ in a study measuring flow through dural puncture sites also measured flow rates through 22-, 26- and 29-gauge needles of two different manufacturers and noted that a long delay in appearance of CSF at the hub may make identification of correct placement in clinical use difficult. The needle-through-needle subarachnoid and epidural technique was described previously and its merits and problems discussed.^{6–10} However, the problem of low flow rates through the long, fine needles has not been addressed.

The results obtained in this study for the standard 26-gauge Becton Dickinson needles were similar to those obtained by Morris *et al.*¹ Palleiko *et al.* found that the new standard Braun Spinocan 26-gauge needle had a resistance-to-fluid flow indistinguishable from that of the BD needle.⁴ This study shows that the resistance-to-fluid flow through the 120-mm Braun 26-gauge Spinocan is over three times greater than that of the 90-mm BD 26-gauge needle. This finding was confirmed by observing the flow rate through the two needles; the time to first drop was over three times greater in the Braun needle compared to the BD needle. Gerrish and Peacock demonstrated that whilst saline was a Newtonian fluid, CSF was not.² Poiseuille's equation for Newtonian fluids under conditions of laminar flow states that the flow rate is directly proportional to the perfusion pressure and the fourth power of the diameter, but inversely proportional to the viscosity of the fluid and the length of the tube. The threefold difference in flow rates between the two needles when this formula is applied to this system, cannot solely be ascribed to the 30-mm difference in length. It would suggest that the internal diameter of the 120-mm needle is less than that of the 90-mm needle, or that the internal finish of the surface is of a poorer quality.

The new Braun Spinocan is a useful addition to the anaesthetists armamentarium. However, it does have a drawback in that although it is one-third longer than the Becton Dickinson needle its resistance is tripled, which

Table 3. Pressure (kPa) and resistance to flow (kPa/(ml/hour)) at 100 ml/hour flow rate.

	Braun needle		BD needle	
	Pressure	Resistance	Pressure	Resistance
Needle number				
1	43.2	0.43	13.2	0.13
2	43.1	0.43	13.8	0.14
3	43.9	0.44	13.2	0.13
4	43.2	0.43	13.2	0.13
5	43.6	0.44	13.3	0.13

leads to increased difficulty in the positive identification of the subarachnoid space by backflow of cerebrospinal fluid. It is important, when using the Braun Spinocan in the needle-through-needle technique, to wait at least 2 minutes for the appearance of CSF in the hub to confirm accurate placement of the needle. The time can be reduced by aspirating on the end of the needle with a syringe, but this adds the hazard of moving the needle from the subarachnoid space by the additional manipulation of the hub. This problem has been circumvented by Nickalls and Dennison who used an artery clip to hold the spinal needle in place.⁸ The hub of the Braun Spinocan is designed to be used in conjunction with Braun Tuohy needles to reduce the risk of this problem so that the two hubs are in close apposition when the tip of the Spinocan is in the subarachnoid space.

Acknowledgments

The author acknowledges the advice given by Dr D. Fell in the preparation of the manuscript.

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Another Selectatec switch malfunction

J. CUDMORE AND J. KEOGH

Summary

Malfunction of a Selectatec-3 switch mechanism is reported which caused isolation of the selected vaporizer. This resulted in a delivery of unsupplemented fresh gas to the patient.

Key words

Equipment; vaporizer.

The Selectatec (Ohmeda) system provides for two vaporizers to be mounted on the back bar of the anaesthetic machine. The system is designed so that the fresh gas flow can pass through only one of the vaporizers. This arrangement prevents the simultaneous administration of two volatile anaesthetic agents and also prevents contamination between vaporizers. A malfunction in the switch mechanism was reported which resulted in complete cessation of gas flow to the patient.¹

Method

The latest malfunction occurred during the course of a busy day-case dental list. It is our practice to use a gaseous induction of anaesthesia with nitrous oxide and oxygen and halothane in suitable patients and to change to isoflurane for maintenance. It was noticed, after the change over to isoflurane, that the anaesthesia became lighter. The machine was checked and no isoflurane could be detected in the fresh gas flow, even though the vaporizer was switched on and the Selectatec switch was set to the correct position. The Selectatec switch appeared to be operating correctly, going from the left to the right position easily, although in retrospect, slightly stiffly.

Anaesthesia was continued with halothane and was completed uneventfully. The switch mechanism was examined by a service engineer who found that both valves had seized in their cylinders because the silicon rubber O-rings which seal the pistons had swelled. The steel ballbearing of the right-hand valve had also worn a track in the softer brass of the pressure plate of the switch mechanism.

The Selectatec switch mechanism contains two spring-loaded ball valve systems which work in such a way that when one is compressed, the other is allowed to cycle to the alternate position. The left-hand valve directs the gas flow to one or other of the vaporizers and the right-hand valve controls the return pathway. The two valves must be cycled in opposite directions to allow the fresh gas to flow correctly through the selected vaporizer (Fig. 1).

The malfunction arose because both valves failed to cycle when the control knob was switched to the right-hand position. This resulted in the position illustrated (Fig. 2), in which the fresh gas was wrongly directed to the left-hand vaporizer and no gas was directed to the right-hand vaporizer. The fresh gas was able to flow freely through the system because the return pathway from the left-hand vaporizer was open.

No difficulty in operating the switch was experienced because as the right-hand valve became stiffer the steel ball bearing of the right-hand valve gradually wore a track in the softer brass of the pressure plate of the switch mechanism so that the gradual increase in pressure required to throw the switch was not noticed.

Discussion

The consequences of this fault depend on the anaesthetic technique in use at the time. Fresh gas without supplemental volatile anaesthetic vapour is directed to the patient so that patients will become lightly anaesthetised and run the risk of 'awareness under anaesthesia' or waking up. The fault described occurred despite the fact that the switch

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Accepted 23 January 1990.

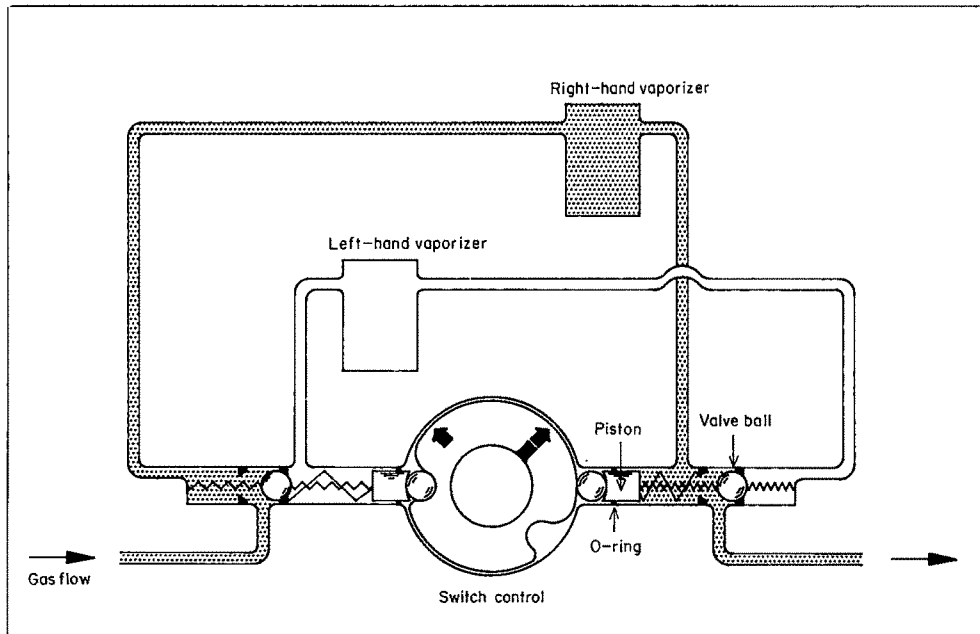


Fig. 1. Diagrammatic representation of the Selectatec switch showing selection of the right-hand vaporizer. Hatching indicates the open pathway.

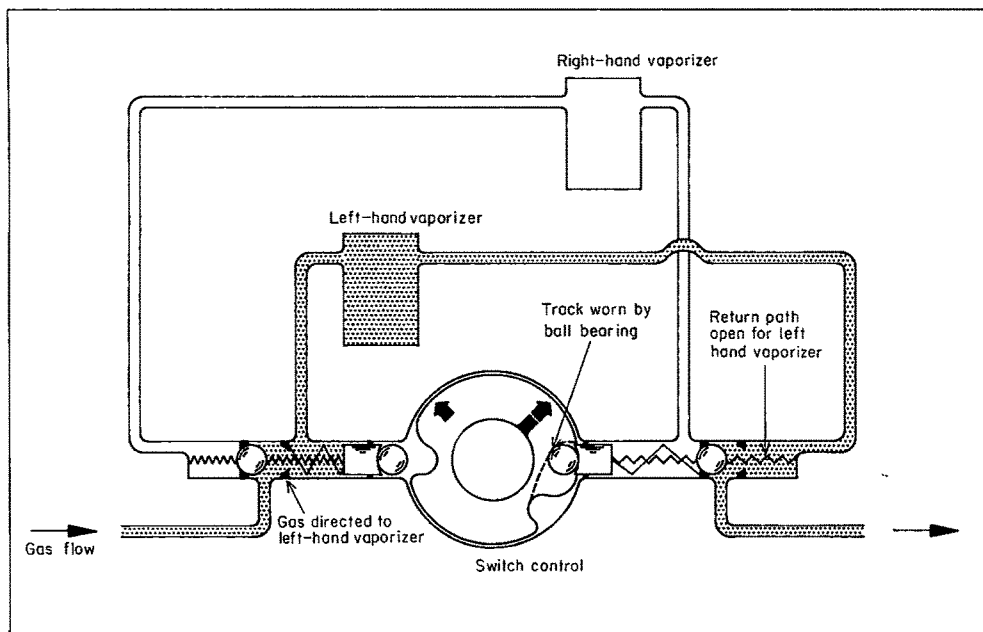


Fig. 2. Diagram illustrates the fault that occurred. Both valves have failed to cycle. The ball-bearing of the right-hand valve moves along the track worn in the pressure plate. Gas flows through the left-hand vaporizer even though the switch is turned to the right-hand position.

could be easily turned in either direction. The only way of detecting the fault is testing for the presence of the vapour in the fresh gas by 'sniffing' or the use of an anaesthetic vapour analyser in the system.

We confirm the misgivings expressed in the previous report with regard to the suitability of such a complex mechanism. The manufacturers changed the design after that report, and lined the barrels of the valves with Teflon. All our Selectatecs were changed to this new design, but are still prone to stick.

It would seem appropriate that, as faults in the mechanism cannot be detected in any other way, the manufacturers shorten their recommended service interval to less than 5

years, and that at that time the switch be dismantled for examination and the o-rings replaced.

Acknowledgments

We thank Mr R. Hale and Mr E. Savage for their help and advice.

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The Trilite inhaler

An historical review and performance assessment

I. E. TWEEDIE AND S. L. SNOWDON

Summary

The Trilite inhaler was developed for use in World War II. Its efficient performance is confirmed and a brief biography of its inventor is also given.

Key words

History; trichloroethylene, inhalers.

The use of trichloroethylene (Trilene, ICI Ltd) for inhalational anaesthesia and analgesia was first described in this country by Dr Langton Hewer at St Bartholomew's Hospital in 1941.¹ Its clinical use was previously described in the USA by Striker 6 years earlier, whilst its use for analgesia in trigeminal neuralgia dates back even further.

The Trilite inhaler is a hand-held Trilene inhaler for inhalational analgesia. It was developed by Dr J.T. Hayward-Butt, with the advice and encouragement of Dr Hewer, for use in the field during World War II. However, it was neither patented nor reported upon until 1947.²

Dr John Terry Hayward-Butt

John Terry Hayward-Butt was born on 31 August 1911. He was educated at Cheltenham College and at Cambridge, graduating MB, BChir from St Bartholomew's in 1937 where he became house officer in ENT surgery, and resident anaesthetist, gaining his DA (Eng) in 1938 and working under Sir Harold Gillies. He had several papers published in the *St Bartholomew's Medical Journal* during this time.

He was anaesthetic registrar at the Royal Free Hospital in 1940 when called up for the navy. His rank had risen to Surgeon Lt.-Commander by the end of the war and it was while he was in the services that he developed his inhaler.

He emigrated to South Africa in 1946 and became Director of the Department of Anaesthesia, and Clinical Tutor at King Edward VIII Hospital, University of Natal, Durban. He published a book, *Trilene analgesia* during 1947 and continued work on the clinical assessment of the inhaler. He stayed in South Africa until 1958; in 1952 he

received his FFARCS and edited the *South African Journal of Anaesthesia*.

He took a post in 1958 as Assistant Professor of Anaesthesiology at the State University of Iowa, USA, where he developed his interest in neuroleptanalgesia, and published a monograph upon the subject. From 1959 until 1971 his work was mainly private practice at the Marieopa General Hospital in Phoenix, Arizona. He then became Associate Professor in Anaesthesiology at the University of Chicago; he had contributed significantly to neuroleptanalgesia, and been one of the pioneers of day-case surgery in America. He died in 1973 aged 62.³

The Trilite inhaler

The Trilite inhaler was a small chrome-plated brass cylinder that measured 21 by 2.2 cm in diameter, and weighed 285 g fully charged; it was portable and robust (Fig. 1). It used air as the carrier gas, and was charged with a 6-ml Trilene ampoule (f), held firmly in position by a spring (b). The ampoule was broken by striking the plunger (e) in the base of the inhaler when analgesia was required. This allowed the Trilene to be absorbed onto a wick (c) wrapped around the vaporizing chamber (g) in the nasal nozzle (d) at the opposite end. The plunger mechanism was integrated with that of the inhalation nozzle in later versions (Fig. 2), but the spent ampoules were still replaced through the base. The patient then inhaled through the Trilite, until analgesia was achieved; this usually required about 20 breaths. Administration was stopped until analgesia was required again, at which time five to six breaths

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Accepted 23 January 1990

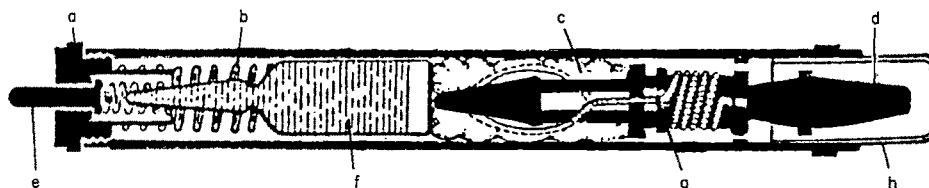


Fig. 1. Sectional details of the Trilite inhaler; (a) closing plug; (b) retaining spring; (c) absorbent pad/wick; (d) nozzle; (e) plunger; (f) Trilene ampoule; (g) capillary wick in vaporizing chamber; (h) protective cap.

were usually adequate.² One Trilene charge lasted 60–90 minutes under these conditions.

There do not appear to be any quantitative performance figures available and so the inhaler was tested in the laboratory, in a manner similar to that described by Dr Hayward-Butt for analgesia.

Method of assessment

The inhaler was set up in a breathing system, so that the experimenter could breathe in through the inspiratory nozzle of the inhaler and out through the adjustable pressure limiting valve. One noticeable feature of the inhaler was its very high resistance to breathing. A 4-litre bag-in-bottle arrangement was interposed between the Trilite inhaler and the subject to prevent the experimenter from breathing the Trilene. The inspiratory tidal volume through the inhaler was measured using a Wright's respirometer, placed on the inhaler side of the bag-in-bottle. This could be seen by the experimenter so that s(he) could monitor her(his) inspiratory tidal volume, keeping it either large (1500 ml) or at a 'resting' tidal volume (650 ml).

The inhaler was placed in a water bath, so that by altering the water temperature, its performance could be assessed at three different temperatures, 10, 20 and 30°C, to simulate a range of field conditions.

The vapour concentration was measured using a Wetenschappelifk Technische Instrumentatie (WTI) Anaesthetic Gas Monitor AG101, which was shown to measure volatile anaesthetic agents accurately.⁴ Calibration was performed using a one-litre gas calibrator containing a 1% v/v Trilene-in-air mixture. Samples of the inspiratory gas were taken from the immediate vicinity of the nozzle. A reference gas was sampled between each cycle of deep and shallow breaths to ensure the absence of any drift in calibration. The results were recorded on a potentiometric chart recorder.

A matrix was set up for measurement of the vapour concentration at different tidal volumes and temperatures. The inhaler was charged with 6 ml of Trilene before use. The inhaler equilibrated with the water temperature for several minutes, then a series of breaths were taken through the apparatus as follows (based on figures given in Dr

Hayward-Butt's paper): 20 × 650 ml breaths, followed by one minute's rest; then three sets of 5 × 650 ml breaths with one minute's rest between each set; 2.5 minute's rest; 20 × 1500 ml breaths, followed by one minute's rest; then three sets of 5 × 1500 ml breaths, at intervals as above. This cycle was repeated at each temperature level.

The results are shown in Table 1. The vapour concentration increased with temperature, but was slightly lower when larger tidal volumes were used (Fig. 3). The concentration in all cases was greater than MAC and in most greater than 2 MAC. It produced a concentration greater than 3 MAC at 30°C during a small tidal volume ventilation, and many of the first peaks recorded were above the upper limit of calibration, that is greater than 1.07% Trilene.

The concentration during an experiment only decreased by approximately 0.03% before the Trilene ran out. The inhaler maintained a satisfactory output for over 200 breaths at the lower temperatures (10–20°C) before the concentration decreased rapidly to below MAC. A marked decrease was observed, however, after only 130 breaths at the higher temperatures. The frequency with which the sets of 5–6 breaths were needed to maintain analgesia is not reported in Dr Hayward-Butt's paper; if, however, one assumes an interval of 2–3 minutes between each set of inhalations from the Trilite then the above figures would be consistent with a single Trilene charge lasting for 60–90 minutes.

Discussion

Dr Hayward-Butt saw the need for an inhaler to administer Trilene as an analgesic under combat conditions, whilst serving in the forces during the war. The criteria he used were that it had to be small, robust, simple to use for self-administration and to require minimal written instructions for non-medical personnel. It had to be ready for immediate use, yet with little risk of loss of the trichloroethylene contained, and allow easy recharge. The original idea came from the successful use of a Trilene-soaked woollen plug in a Benzadrine inhaler, to give brief inhalational analgesia to injured assault troops.

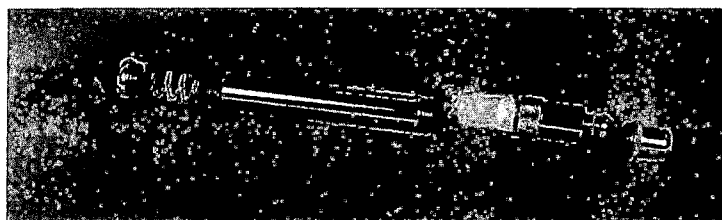


Fig. 2. Trilite inhaler showing later modification of a combined nozzle/plunger.

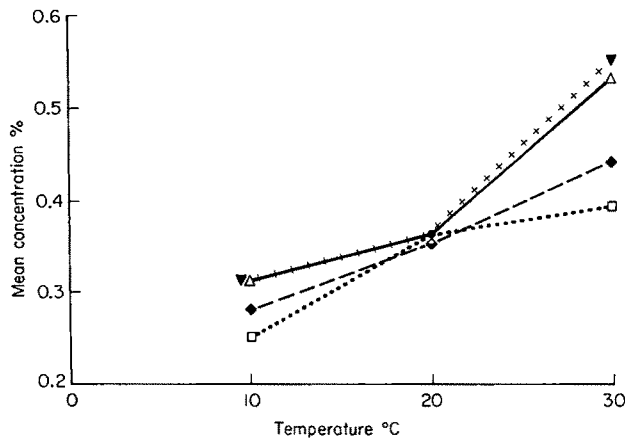


Fig. 3. Mean concentration against temperature for the different depths and number of breaths. ∇ — ∇ , 5 \times 650 ml breaths; \triangle — \triangle , 20 \times 650 ml breaths; \blacklozenge — \blacklozenge , 5 \times 1500 ml breaths; \square — \square , 20 \times 1500 ml breaths.

Dr Hayward-Butt validated the Trilite inhaler's clinical effectiveness for analgesia during minor surgical and obstetric procedures on 1183 patients in Britain and South Africa when the war ended in 1945. It proved successful in the majority of patients (85%).² Hill and Calvert, at the time, claimed similar results for successful analgesia using Trilene self-administration apparatus of their own design, for minor surgery and obstetrics.^{5,6} Barratt and Platt in a review of Trilene use in general practice also had similar success rates.⁷ Hill published performance figures for his apparatus, and stated it produced an approximate Trilene concentration of 2.4–4.8%. However, his apparatus was designed on a plenum-like concept, which may explain the higher concentrations.

Our observations support the efficacy claimed for the Trilite when consideration is given to the MAC value of 0.17% for trichloroethylene and the volume percent concentration, 0.35–0.5%, delivered by Tecota and Emotril inhalers when used intermittently and which give satisfactory analgesia. The Trilite inhaler thus appears to have been a simple, robust and effective method of administering Trilene analgesia.

Table 1. Trichloroethylene concentrations showing the mean steady state values and ranges.

Water bath temperature (°C)	Tidal volume (ml)	Number of breaths	Mean concentrations steady state (%)	Range (%)
10	560	20	0.31	0.3–0.31
		5	0.31	
	1500	20	0.25	0.25–0.29
		5	0.28	
20	650	20	0.36	0.31–0.41
		5	0.36	
	1500	20	0.36	0.32–0.37
		5	0.36	
30	650	20	0.53	0.53–0.56
		5	0.55	
	1500	20	0.39	0.39–0.44
		5	0.44	

Acknowledgments

We thank Mr Monchard who found the inhaler in his dental surgery and brought it to our attention. Our gratitude also goes to Dr N.E. Williams who has given much encouragement.

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Forum

The laryngeal mask airway in children

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Summary

The laryngeal mask airway was used in 200 children during a variety of surgical procedures. Some problem with the use of the device was encountered in 47 cases (23%), but in only five cases (2.5%) were the problems serious enough to warrant abandonment of its use. A clear airway was ultimately achieved in 191 children. Downfolding of the epiglottis over the laryngeal inlet was identified in eight out of 24 patients where flexible laryngoscopy was performed, clinically all these had unobstructed airways. The mask was used in 16 children with known airway problems. It is concluded that the size 2 laryngeal mask airway can be successfully used within the weight range 6–30 kg.

Key words

Anaesthesia; paediatric.

Equipment; laryngeal mask airway.

The laryngeal mask airway (LMA) is the development of a new concept in upper airway management. Its inception was a result of the application of bio-engineering and the postmortem examination of the adult larynx.¹ This led to the creation of a prototype mask, which has been the subject of independent studies in spontaneously breathing adults.^{1–3}

A survey of the use of the commercially available LMA over one month in a district general hospital indicates the rapid growth in the use of the device in adult anaesthetic practice.⁴ It appears to be relatively simple and safe to use across a wide range of surgical specialties.³ It has also been advocated for use in the management of the difficult airway.^{5–7} The device does not, however, provide a water-tight seal around the larynx, and should not be used in patients at risk of regurgitation. There is a risk of gastric inflation during positive pressure ventilation.

The LMA is now available for use in children in two sizes. They are scaled-down versions of the adult forms and no direct postmortem specimen work has been performed. The infant and young child have a relatively large tongue in relation to the mandible, the glottis lies higher and more anteriorly than in the adult, while the vocal cords are angled more forwards and downwards. The epiglottis is large and floppy, and may lie against the posterior wall of the pharynx which can cause upper airway obstruction during anaesthesia. We performed a clinical evaluation in paediatric anaesthesia in view of the above differences and since the use of the LMA in children is becoming increasingly common.

Method

The size 1 and 2 LMAs (which the manufacturers recommend for use in children who weigh < 6.5 kg and 6.5 to 25 kg respectively) were made available to all anaesthetists working in the department. Basic guidelines were given in

its use before the start of the study. The anaesthetic staff were free to use the device at their discretion.

Premedication was given in almost all cases and consisted of an anticholinergic with or without an opioid or sedative drug. Induction of anaesthesia was with either 50% cyclopropane in oxygen or intravenous thiopentone. Anaesthesia was maintained using halothane, or occasionally isoflurane, in nitrous oxide and oxygen; the patients were allowed to breathe spontaneously through a T-piece system. Monitoring of arterial oxygen saturation, ECG and blood pressure was then started. The LMA was inserted when anaesthesia was adequate, the mask inflated with 10 ml air and connected to the T-piece system via a 15-mm Portex connector. It was then fixed in place using one inch Elastoplast. The device was left *in situ* after surgery until the return of laryngeal reflexes.

A questionnaire was completed if the device was used with this information: age, weight and any pre-existing airway problems; grade of anaesthetist and previous experience with the mask; the operation and duration of insertion; the ease of insertion, the number of attempts and any associated problems; quality of the airway and manoeuvres necessary to achieve a perfect airway; presence of a leak on compression of the reservoir bag; the ease of removal and any associated problems.

The only exclusion criteria were patients at risk of regurgitation of gastric contents. The position of the mask was verified as previously described.^{1–3} In addition, in some cases, flexible laryngoscopy was performed through the LMA. The device was washed with soap and water on removal, and boiled for 3 minutes before re-use.

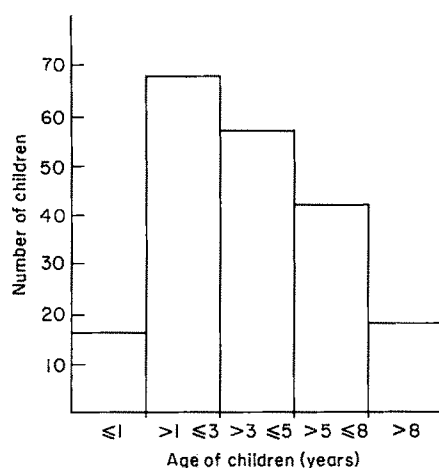
Results

The size 2 LMA was used in 200 children during a period of 3 months. Table 1 give the details of the demographic data and duration of insertion of the mask and shows that

Accepted 17 January 1990.

Table 1. Demographic details and duration of insertion.

	Median	Range
Age (years)	3.7	0.5–11.9
Weight (kg)	15.2	5.7–45.0
Duration (minutes)	30	7.0–90.0

**Fig. 1.** Age distribution of children.**Table 2.** Problems with the use of the LMA related to previous experience.

Previous experience (Number of cases)	Number of problems
≤ 5	18/56 (32%)
> 5 ≤ 10	10/27 (37%)
> 10 ≤ 15	3/18 (17%)
> 15 ≤ 20	1/10 (10%)
> 20	15/89 (17%)

Table 3. Operative procedures.

	Number %
General surgery	75 (37.5)
Orthopaedics	44 (22)
Plastic surgery	21 (10.5)
ENT/dental	18 (9.0)
Radiology	13 (6.5)
Urology	13 (6.5)
Ophthalmology	11 (5.5)
Other	5 (2.5)

Table 4. Problems related to insertion and removal.

Insertion	Number	Removal	Number
Difficulty in negotiating posterior pharynx	10	Coughing	12
Coughing	5	Bitting	11
Laryngospasm	4	Laryngospasm	5
Desaturation > 5%	3	Retching	4
Breath holding	2	Vomiting	3
Vomiting	1	Other	2
Excess saliva	1		

Table 5. Manoeuvres used to relieve airway obstruction.

	Number
Remove and reinsert	13
Reposition head	6
Add more air to mask	5
Remove air from mask	2
Apply CPAP	1
Abandon	5

the device was used successfully at weights above and below those recommended. The age distribution of the children is shown in Figure 1.

Sixteen anaesthetists participated in the study. Six were consultants who between them inserted 74 masks, four were senior registrars who inserted 28 masks and the remaining 98 masks were inserted by six registrars. The number of problems encountered related to previous experience with the device is shown in Table 2. It was used across a large range of surgical specialties (Table 3), but most frequently for general surgical and orthopaedic procedures.

Some problem with the use of the device was encountered in 47 cases (23%), but in only five cases (2.5%) were the problems serious enough to warrant abandonment of its use. It was easy to insert in 185 (92.5%) cases. Problems occurred in 22 patients immediately after insertion (Table 4). Two of these patients had multiple problems and the use of the device was abandoned.

The LMA was correctly inserted on the first attempt in 179 (89.5%) cases. Of the 21 remaining, two were then abandoned and of the other 19, 16 were successfully inserted on the second attempt. None of the remaining three were successfully inserted despite further attempts.

One hundred and seventy-eight children had a completely clear airway after successful insertion. A clear airway was ultimately achieved after a variety of manoeuvres (Table 5) in 191 children. Four of the remaining nine patients had partial obstruction but the use of the device was continued and five were abandoned for a number of reasons. A 5-year-old child had previous subglottic stenosis and despite three attempts at insertion, an unobstructed airway could not be achieved; subsequent tracheal intubation was performed uneventfully. Two others aged 22 months and 9 years had no known airway problems, but coughing, laryngospasm and vomiting occurred after insertion. Insufficient anaesthesia was responsible in both cases. In addition, excessive secretions from lack of premedication aggravated the situation in the second child. Complete airway obstruction occurred in the two remaining children, aged 2 and 5.5 years, despite three attempts at insertion. An oropharyngeal airway and face-mask were subsequently employed.

A leak was audible in 52% of cases on gentle manual compression of the reservoir bag. No formal attempt to quantify the degree of leak was made. No further problems were reported until the recovery period, once the device was successfully inserted and a clear airway achieved.

The LMA was removed with ease in 191 children (95.5%), but problems associated with removal occurred in 26 cases (Table 4). Single problems occurred in 19, while seven had multiple problems. No child required active intervention, other than removal of the device, as a result of problems on recovery.

In 24 patients direct laryngoscopic views of the laryngeal inlet were obtained using a flexible fiberoptic bronchoscope. In 16 of these endoscopies the vocal cords could be clearly seen, while in eight downfolding of the epiglottis



Fig. 2. Photographic views as seen through the LMA of the laryngeal inlet. The vocal cords and arytenoids can be seen clearly on the left, while downfolding of the epiglottis beneath the fenestrations of the mask aperture has occurred on the right.

Table 6. Known airway problems.

	Comments
Subglottic stenosis	Partial obstruction abandoned after three attempts of insertion
Amyoplasia congenita	No problems
Apert's syndrome, five cases	Four had no problems. One had excess secretions and required two attempts of insertion
Cleft palate, two cases	One had laryngospasm and required two attempts of insertion
Tracheomalacia, post aortopexy	Had laryngospasm and required two attempts of insertion
Haemangioma of pharynx and larynx	No problems
Multiple fibromatosis	No problems
Sleep apnoea upper airway obstruction	No problems
Haemangioma on lip	Required two attempts of insertion
Hydrocephalus and achondroplasia	No problems
Other	Partial obstruction

beneath fenestrations of the LMA aperture was observed (Fig. 2).

Airway problems existed in 16 patients before the use of the LMA (Table 6). One case was abandoned as described above, one had partial airway obstruction and four required more than one attempt at insertion to achieve a satisfactory airway.

The size 1 mask was only used on two occasions.

Discussion

The various merits and limitations of the LMA in adults have been described.³ Our incidence of major problems that resulted in abandonment of its use (2.5%) is comparable with that reported by others.^{3,4} Minor problems, however, occurred more frequently in our study. Inadequate anaesthesia on insertion of the LMA may result in coughing, vomiting, and laryngospasm. Thus sufficient anaesthesia is essential and this appears to be greater than that required for insertion of an oropharyngeal airway, but less than that for tracheal intubation.

In some cases, particularly in children with tonsillar hypertrophy, insertion can prove difficult, but can be facilitated by ensuring that an antisialogue premedicant has been given, that the head is well extended, and that firm pressure of the device on the hard palate is applied in order to negotiate the posterior pharyngeal wall. Other manoeuvres can be employed to overcome this obstacle, namely by slight lateral insertion of the mask, by the use of a finger on the posterior wall of the mask acting as a guide, and by pulling the tongue forwards with a dry swab.

Once inserted, accidental dislodgement can occur, especially in the younger child, where there is a substantial portion of the tube protruding from the mouth. Thus it is important to fix the LMA securely in place. Biting on the tube of the LMA during recovery can cause airway obstruction. This can be overcome by the use of a bite block such as Guedel airway which should be inserted at the same time as the LMA and also securely fixed in place.

The device does not provide an absolute guarantee of a secure airway, or of laryngeal protection, since it does not provide a watertight seal and thus aspiration may occur in patients who are at risk of regurgitation of gastric contents. Similarly, many of the problems encountered on recovery appeared to be due to accumulation of secretions within the mask aperture.

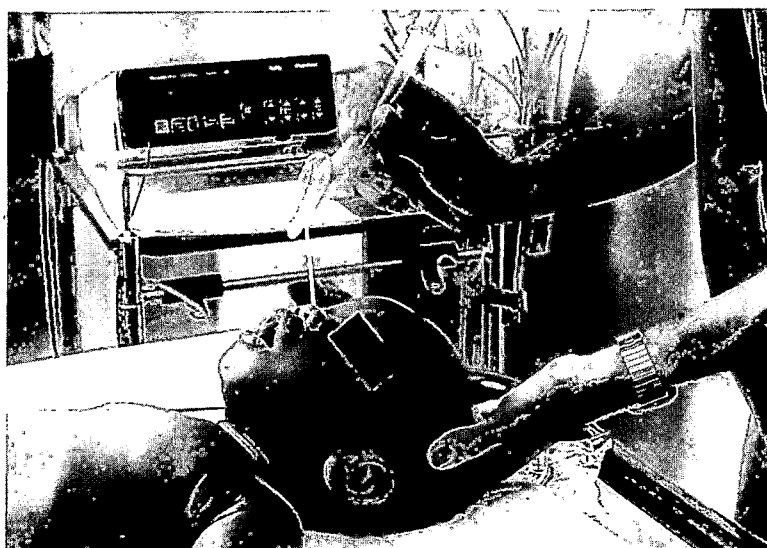


Fig. 3. The size 2 laryngeal mask airway.

Intermittent positive pressure ventilation via the LMA could potentially result in gastric inflation which, in children, will cause splinting of the diaphragm and compromise respiratory function. The lungs of two of the patients in the study were ventilated successfully, but further work will need to be performed to validate this technique in children.

The LMA was used in several children with known difficult airways, but problems did occur. Hence great caution should be taken with these patients. It is important that alternative means to maintain an airway are available and that there is ready access to the patient's head. We would not regard the device as the technique of choice for patients with known airway problems presenting for major head and neck surgery.

We were interested to note that downfolding of the epiglottis does occur as feared in some cases. Surprisingly, this did not result in airway obstruction. Presumably this is because the area between the edges of the epiglottis and the rim of the mask aperture is sufficiently large to allow unobstructed ventilation.

The LMA proved particularly suitable for the spontaneously breathing child as an alternative to the use of a mask and airway or tracheal tube. It maintained a clear airway without the need for laryngoscopy and intubation and allowed both hands to be free. The size-2 LMA was remarkably versatile in that it could be used over a wide age range. The mask was well tolerated once in place, even at light planes of anaesthesia.

Airway maintenance during recovery was facilitated. The device was removed once the patients were awake and had full return of their laryngeal reflexes. Connexion directly to a T-piece system provided an additional monitor of respiration whilst 100% oxygen was administered. As in adults, the LMA was particularly useful for minor head and neck procedures such as myringotomy and for peripheral limb, minor general and urogenital surgery in conjunction with a local anaesthetic block.

We consider that the LMA provides the anaesthetist with

an additional aid in the management of the upper airway in children during spontaneous breathing. An adequate period of starvation is essential before its use, and an antisialogue premedicant should be given. Any method of induction is suitable for its insertion in children, but an adequate depth of anaesthesia is necessary. Secure fixation is important to prevent accidental dislodgement, and the use of an oropharyngeal airway one size smaller than for the age of the child acts as a good bite block. The size-2 LMA can be successfully used within a weight range of between 6 kg to 30 kg.

Acknowledgments

The authors thank Dr I.G. Johnston and all the members of the anaesthetic department at the Hospital for Sick Children, Great Ormond Street for their assistance during this study and Dr J.S. Sherrard for her help in text preparation.

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Anaesthesia, 1990, Volume 45, pages 763–766

Intravenous diclofenac sodium

Does its administration before operation suppress postoperative pain?

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Summary

Intravenous diclofenac sodium was evaluated in a double-blind randomised trial relative to intramuscular diclofenac, intravenous fentanyl, and intramuscular placebo in 160 patients undergoing extraction of impacted lower third molar teeth. The test drug was administered before operation in an attempt to alleviate postoperative pain. A 10-cm visual analogue scale was used to assess pain at 30 minutes and one day after surgery, if the patients stayed overnight. Patients who received intravenous diclofenac had significantly less pain than the other groups 30 minutes after operation. They also had significantly less pain one day after surgery than the placebo or opioid groups, but not less than the intramuscular diclofenac group. Capillary bleeding time, in comparison with placebo, was significantly prolonged after the use of intramuscular diclofenac, and a similar but nonsignificant trend was observed in the intravenous diclofenac group. No problems were encountered with excessive bleeding in any group.

Key words:

*Analgesics; diclofenac, fentanyl.
Pain; postoperative.*

Accepted 23 January 1990.

The management of moderate postoperative pain can be a problem since analgesics such as paracetamol may not be sufficiently potent. Opioids are good analgesics but frequently only a quarter of the prescribed dose is given, which results in a poor quality of analgesia.¹ In addition nausea and dizziness may follow opioid administration and can occasionally be quite severe and incapacitating.²

McQuay and his colleagues³ have demonstrated that an effective reduction in postoperative pain can be achieved if an analgesic is given before surgery. In the absence of regional analgesia or opioids, surgery results in an afferent bombardment of the central nervous system (CNS) with resultant hyperexcitability of the spinal cord. The administration of analgesics before operation reduces this CNS barrage and prolonged cord hyperexcitability.⁴ Prostaglandin inhibitors have been successfully used to inhibit some types of postoperative pain and reduce opioid requirements in others.^{5,6} These drugs work at a peripheral level to prevent prostaglandin synthesis when tissues are damaged, and result in a reduction of free nerve ending sensitisation.^{7,8}

Antiprostaglandin activity may be ineffective unless the preparations are given at the appropriate time before surgery. The timing of drug administration can be difficult if an operating list does not run to schedule. In an attempt to minimise these problems, the benefit derived from the intravenous administration of the nonsteroidal anti-inflammatory drug (NSAID) diclofenac sodium was explored in patients admitted for extraction of impacted lower third molars. The study aimed to determine if the intravenous administration of diclofenac sodium immediately before surgery would markedly reduce pain in the postoperative period. Comparison was made with intramuscular diclofenac, intravenous fentanyl and an inert preparation. NSAIDs have aspirin-like activity so observations were also made on platelet activity by noting the bleeding time before and after test drug administration.

Methods

One hundred and sixty fit (ASA 1) patients aged between 16 and 65 years admitted for extraction of two lower third molar teeth were studied. Participants gave verbal consent for the study which was approved by the Research Ethics Committee, Queen's University, Belfast.

Premedication was with diazepam 10 mg. Patients were randomly assigned to receive one of the following on a double blind basis, immediately after induction of anaesthesia: intramuscular normal saline (maximum 3 ml); intramuscular diclofenac sodium 1 mg/kg (maximum 75 mg in 3 ml); intravenous diclofenac sodium 1 mg/kg (maximum 75 mg in 18 ml); intravenous fentanyl 1 µg/kg (maximum 100 µg in 18 ml). Observations were carried out by one of two recovery ward nurses who were unaware of the treatment received but who had been instructed on the method of administering the visual analogue scale (VAS).

Induction of anaesthesia was with propofol 2 mg/kg, gallamine 20 mg followed by suxamethonium 1 mg/kg to facilitate tracheal intubation, and anaesthesia was continued with halothane and oxygen in 60% nitrous oxide. Co-codaprin tablets (dispersible aspirin and codeine) and intramuscular levorphanol were available to all groups, as deemed necessary by the two recovery ward nurses. Co-codaprin alone was also available to all patients on a '4-hourly, as required' basis in their own wards.

A 10-cm VAS was used to determine pre-operative anxiety and expected pain in the anaesthetic room (anchor points were 'totally relaxed—extremely anxious', and 'no pain—worst possible pain' respectively). Perceived pain was estimated on a VAS approximately 30 minutes after

Table 1. Grading of surgical difficulty.

I	Simple elevation without bone removal.
II	Simple elevation after minimal bone removal.
III	Wide bone removal or tooth section.
IV	Wide bone removal and tooth section.

completion of surgery (by nurses) and again at 1000 hours the following day (by one of two housemen trained in using VAS). Day patients did not complete a VAS for pain on the day after surgery. Analgesic requirements were also recorded. On completion of the procedure the surgical difficulty was rated from I to IV (Table 1).

The capillary bleeding time was measured in 15 randomly selected subjects from each group of 40 immediately after induction of anaesthesia. Measurement was carried out by a technique similar to that described by Ivy.⁹ A tourniquet was inflated to 40 mmHg and three skin punctures were made over the lateral aspect of the forearm using an Autolet device. The mean time taken for bleeding to cease, when the blood drop was absorbed by filter paper every 15 seconds was recorded. This procedure was carried out before and approximately 20 minutes after the test drug was administered.

Comparability of the groups was assessed using one-way analysis of variance or Chi-squared tests for contingency tables as appropriate. VAS results (after arcsin transformation) and changes in bleeding time were compared using one-way analysis of variance, followed by the Student-Newman-Keuls multiple range test if a significant F statistic was obtained. Observations on analgesic requirements were analysed using Chi-squared tests. The 5% level of significance was used throughout.

Results

The groups were broadly similar with respect to age, sex, weight, duration of operation and difficulty of surgery (Table 2). The differences in mean age between the groups attained significance, although this was not considered likely to bias the results. No significant difference was detected between the groups as regards pre-operative anxiety, expected pain and pre-operative bleeding time.

Observations on perceived pain using a VAS at 30 minutes postoperatively (Table 3) indicated that the intravenous diclofenac group suffered significantly less pain than patients in all other groups ($p < 0.05$). In those patients (31–37 in each group, who stayed in hospital overnight) pain scores the next morning were markedly lower in the intravenous diclofenac group relative to the placebo and fentanyl groups ($p < 0.05$). Many of the patients in the intravenous diclofenac group did not require any postoperative analgesia. This was significant compared to the fentanyl and placebo groups ($p = 0.006$ and $p = 0.03$ respectively). A similar but nonsignificant trend was noted in the intramuscular diclofenac group.

Compared with the control readings, bleeding time was only significantly increased in those patients given intravenous diclofenac (Table 4, $p < 0.05$ compared with placebo).

Discussion

The release of prostaglandins during tissue damage is considered to enhance the action of bradykinin on nociceptors¹⁰ and hence accentuate nociception.^{11,12} Aspirin-like drugs are generally considered to be weak analgesics but their ability to inhibit prostaglandin synthesis¹³ can result in very effective analgesia in certain situations. NSAIDs, when given before tissue damage may prevent nociceptor

Table 2. Characteristics of the four groups. Mean (SD), frequency.

	Intramuscular saline	Intramuscular diclofenac	Intravenous diclofenac	Intravenous fentanyl
Difficulty I	4	3	1	2
II	22	17	18	22
III	9	16	16	14
IV	5	4	5	2
Mean age; years	23.4 (4.4)	25.8 (5.1)	25.4 (7.2)	28.5 (10.3)
Sex M:F	13:27	20:20	21:19	13:27
Mean weight; kg	65.4 (11.9)	66.5 (12.2)	68.3 (13.3)	63.0 (9.1)
Expected pain	55.3 (19.8)	52.3 (22.7)	48.4 (21.6)	51.3 (20.9)
Anxiety	56.6 (19.8)	52.8 (24.0)	46.6 (22.4)	50.6 (21.2)
Mean duration of operation (minutes)	34.0 (10.7)	36.3 (12.1)	37.3 (11.9)	33.9 (12.4)

Table 3. Postoperative observations. Mean (SD), frequency.

	Intramuscular saline (n = 40)	Intramuscular diclofenac (n = 40)	Intravenous diclofenac (n = 40)	Intravenous fentanyl (n = 40)
Pain severity at 30 minutes	52.2 (22.9)	45.0 (19.8)	33.4 (17.3)	50.3 (20.5)
Analgesic needs				
Nil	5	12	14	3
Co-codaprin	29	24	24	26
Levorphanol	6	4	2	11
Pain severity on first postoperative day	21.5 (18.0)	15.9 (12.7)	12.7 (12.2)	22.3 (11.2)
n	33	31	37	35

Table 4. Pre-operative ($t = 0$ minutes), intra-operative ($t = 20$ minutes) and changes in bleeding times (seconds); mean (SD).

	Intramuscular saline	Intramuscular diclofenac	Intravenous diclofenac	Intravenous fentanyl
Pre-operative bleeding time	140.7 (37.1)	131.3 (38.1)	132.7 (43.3)	129.3 (28.5)
Intra-operative bleeding time	111.7 (32.2)	140.6 (37.9)	127.7 (20.3)	112.6 (29.9)
Change in bleeding time	-29.0 (33.9)	+9.3 (30.5)	-5.0 (36.7)	-16.7 (24.3)

sensitisation and possibly reduce the CNS bombardment described by Wall.^{4,12}

The intravenous administration of a NSAID enables high tissue levels to be achieved within a few minutes. The intramuscular administration of diclofenac sodium takes approximately 0.5 hours to achieve peak plasma levels.¹⁴ This corresponds to the duration of surgery so that one would expect optimum analgesia at this time. However, intravenous diclofenac sodium provided better post-operative analgesia despite the fact that plasma levels would have decreased rapidly in the preceding 30 minutes. This would indicate that inhibition of prostaglandin synthesis before operation provides better protection against peripheral nerve sensitisation, than administration after tissue disruption.

We chose to observe the effects of an opioid with a relatively short duration of action (fentanyl), in addition to placebo; the purpose was to determine whether this opioid could reduce CNS afferent bombardment during surgery and thus reduce postoperative CNS hyperactivity and post-operative pain.⁴ The fentanyl should have acted over the 20–30 minutes of surgery, although it did not significantly reduce postoperative pain. This may have been because of an inadequate dose of fentanyl; however, a larger dose might have caused respiratory depression in these spontaneously breathing patients. An opioid with a long duration of action was not used since undesirable side effects might have delayed recovery, especially in the day stay cases.

Pain intensity was assessed using VAS since these are considered to be reliable and capable of fine resolution.¹⁵ In addition, patients could readily understand them during the early recovery phase, since they had experience in their use just before anaesthetic induction. A record of analgesic use was kept, although this alone was not considered a sensitive measure of pain intensity, since the administration of oral or intramuscular preparations depends on the nurse's interpretation of pain intensity and the patient's ability to communicate this. This problem does not exist where patient-controlled analgesic devices are used. The measure of simple analgesic consumption correlates with trait-anxiety and therefore provides a poor assessment of pain intensity.¹⁶ The greatest number of patients not requiring any postoperative analgesia were within the intramuscular and intravenous diclofenac groups despite these limitations. The lowest VAS scores were also within these groups.

The intravenous administration of the intramuscular diclofenac preparation results in a high incidence of venous thrombosis unless diluted beforehand.¹⁷ Patients with renal disease were excluded from the study since NSAIDs are known to inhibit the vasodilator prostaglandins synthesised in the kidney. The administration of a NSAID results in a decrease in renal plasma flow in the short term, although no significant or clinically important changes have been observed in the long-term, in healthy volunteers.¹⁸

Intravenous diclofenac has been safely used in clinical^{19,20}

as well as in pharmacokinetic studies in man.¹⁴ However, the intravenous route of administration is not recommended by the manufacturers at present simply because there is inadequate information (Ciba-Geigy Pharmaceuticals, personal communication). The mean terminal half-life in plasma is 1.1 hours following intravenous administration and it appears to obey 3-compartment open model kinetics; elimination occurs from the central compartment.²¹ The short biological half-life of diclofenac suggests that one or more of the active metabolites may remain in the body for longer than the parent drug, to give rise to a prolonged clinical effect. This effect might give rise to some postoperative analgesia but would hardly explain why VAS ratings were so good the day after surgery.

Excessive bleeding did not occur in any case studied. However, the bleeding times which were carried out indicated some antiplatelet activity because of diclofenac sodium. This is in keeping with other work, which suggests that bleeding and coagulation disturbances are not so marked with NSAIDs as with aspirin.^{22,23} Aspirin blocks cyclo-oxygenase irreversibly, resulting in ineffective thrombocyte aggregation during their lifetime. Diclofenac causes a reversible block, and only alters bleeding time during the activity of the drug.²⁴ It is interesting that the intramuscular diclofenac altered bleeding time more than intravenous diclofenac. This may have been the result of greater diclofenac activity from the intramuscular route at 20 minutes when the second bleeding time was carried out, than from the intravenous route after this time.

Diclofenac administered before oral surgery provided better postoperative analgesia than did fentanyl. The intravenous route allowed better control of blood levels of the drug in use. Surgical or postoperative bleeding was not a problem after either intramuscular or intravenous administration of any of the drugs tested. The intravenous administration of diclofenac sodium may also obtund pain if given before other body surface operations.

Acknowledgments

The authors are indebted to the recovery ward nurses who administered the VAS and the dental housemen who administered the VAS the day after surgery. We are also very grateful to the Royal Victoria Hospital Pharmacy Production Unit, who formulated preparations for this study, Miss C. Davidson who typed the manuscript and Ciba-Geigy Pharmaceuticals who provided technical and financial assistance.

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An evaluation of a 30-gauge needle for spinal anaesthesia for Caesarean section

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Summary

A 30-gauge spinal needle was evaluated for Caesarean section, using a combined epidural/spinal technique, in 50 mothers. Spinal anaesthesia failed in six mothers and was inadequate in another six. General anaesthesia was required on one occasion. A 25% overall failure rate suggests that a 30-gauge needle is not a practical proposition for routine clinical practice.

Key words

Anaesthesia; obstetric.

Anaesthetic techniques, regional; spinal.

Spinal anaesthesia for Caesarean section has not achieved the same popularity as epidural anaesthesia despite its superiority in terms of speed of onset¹ and density of analgesia.² Many anaesthetists believe that the incidence of headache in the obstetric patient when using the 25-gauge needle³ is unacceptable. An audit of 91 spinal anaesthetics performed in our obstetric unit using 26-gauge needles in 1988 showed that two patients had required epidural blood patch, while no severe headaches were reported after 1781 epidurals administered over the same period. The advantages of combining both techniques for anaesthesia for Caesarean section have been argued elsewhere,² but are diminished with even a small incidence of severe headache attached to the spinal element.

If hypotension caused by the precipitate action of intrathecal local anaesthetics can be avoided, then a combination of techniques will only yield the best of both worlds if spinal headache can be prevented. This study, therefore, evaluates a 30-gauge spinal needle used in conjunction with a spinal/epidural technique for Caesarean section. The 30-gauge needle was chosen for evaluation since it was the finest available to us, and we anticipated that its use would abolish spinal headache.

Method

Permission for the study was given by the local ethics committee. Fifty mothers who requested regional anaesthesia for Caesarean section were recruited and informed consent obtained. All mothers were prepared as for general anaesthesia. Fluid loading was carried out using both gelatin solution (Haemaccel) and 0.9% saline to a maximum of 1500 ml. Patients were draped in the right lateral position, and an epidural sited in either the L_{2/3} or L_{3/4} interspace, using loss of resistance to saline. Once the Tuohy needle was correctly positioned, an 11-cm 30-gauge needle was passed down, and, by virtue of its double Luer hub, was locked into the Tuohy. Heavy bupivacaine 1.5 to 2.0 ml was then injected, whether or not cerebrospinal fluid (CSF) was visible. The 30-gauge needle was then removed and replaced with an epidural catheter, the Tuohy needle was removed and a dressing applied. The mother was immediately turned into the supine position and tilted to the left in preparation for surgery.

Height of analgesia was assessed using ice and pinprick every minute for 5 minutes, and then every 5 minutes for the next 30 minutes. A record of arterial blood pressure and heart rate (Dinamap, Critikon) was made at the same time. Ephedrine requirement, analgesic supplements and epidural top-ups were also noted, as were time taken in the anaesthetic room, maternal complications, and Apgar scores.

All mothers received 3 mg preservative-free morphine administered epidurally at the end of the operation.

Postoperative assessment included overall impression (good/fair/poor), the degree of analgesia obtained (good/fair/poor), and impression of motor block (pleasant/not bothered/unpleasant). Patients were questioned about headache, paraesthesia, weakness, backache, urinary problems and mobilisation, and were asked to make comparisons with previous anaesthetics, where appropriate.

Results

The mean age of the mothers was 26 years (SD 5.2), height 158 cm (SD 9.0) and weight 75 kg (SD 17.0).

Efficacy of block. Spinal anaesthesia alone was adequate in 37 (74%) mothers. In 12 (24%), spinal anaesthesia failed; the block was either inadequate or a complete failure and epidural anaesthesia with either bupivacaine or lignocaine was uneventful. One mother received a general anaesthetic after spinal anaesthesia had failed.

Analgesic supplements. One mother (2%) in the spinal group, and six (50%) in the epidural group required supplements of either alfentanil or propofol.

Height of block. Within 5 minutes of injection of intrathecal bupivacaine, the height of analgesia had reached T₆. The ultimate height of successful blocks was between T₂ and T₆.

Hypotension. Fourteen (38%) mothers in the spinal group, and two (17%) in the epidural group recorded a systolic arterial blood pressure below 90 mmHg in the first 15 minutes. Thirty three (89%) mothers in the spinal group and eight (67%) in the epidural group required ephedrine.

Time in anaesthetic room. The time taken from gowning and injecting to appearing in the operating theatre were respectively 20 and 12 minutes for the spinal group, and 30 and 21 minutes for the epidural group (Table 1). The

Table 1. Times in minutes (mean, SD) from gowning, and from the injection of local anaesthetic, to transferring the patient to theatre.

	All patients (n = 49)	Spinal (n = 37)	Epidural (n = 12)
Gloves to theatre	23	20 (7.96)	30 (7.5)
Injection to theatre	13	12 (4.58)	21 (4.52)

Table 2. The incidence of peri-operative side effects.

	All patients (n = 49)	Spinal (n = 37)	Epidural (n = 12)
Nausea/vomiting	14	7	7
Poor analgesia	9	1	8
Dizziness	3	0	3
Heavy arms	2	1	1
Shivering	1	0	1
Sedation	1	0	1

incidences of intra-operative problems are shown in Table 2.

Postoperative assessment. On direct questioning four (11%) patients in the spinal group admitted to a mild and transient headache, one of these had had two suspected dural punctures. Two (5%) patients in the spinal group had persistent analgesia. Five (13%) patients in the spinal group and eight (70%) patients in the epidural group complained of backache.

Patients impressions. Eleven out of 15 with a previous epidural anaesthetic preferred spinal anaesthesia, while eight out of 10 who had a previous general anaesthetic preferred spinal anaesthesia. The overall impression in the spinal group was good in 81% of patients, fair in 16% and poor in 3%. Motor block was thought to be unpleasant in 16% of mothers, but 84% were not bothered.

Discussion

The relatively high incidence of postspinal headache in the obstetric patient is considered to be a major disadvantage of the technique. Significant benefit should accrue from using a fine needle if leakage of CSF through the dural puncture is the cause of the headache. Somewhere, however, a balance has to be struck between practicality, on the one hand, and a reduced incidence of side effects on the other. The 30-gauge needle was chosen because we believed that it would effectively probe the nature of this balance.

The advantages of a combined spinal/epidural anaesthetic for Caesarean section have been debated elsewhere.² It was considered to be particularly suitable for a fine spinal needle since the Tuohy could act as an introducer, and the need for a single skin puncture bestowed an additional elegance on the technique. However, brisk action at the end of the intrathecal injection is required if

an epidural catheter is to be placed before the local anaesthetic has fixed. A problem with catheter placement, for example intravenous siting, is likely to result in inadequate spread of anaesthetic. We believed that a test dose was worthwhile to test for inadvertent intravenous placement of the catheter.

A 30-gauge spinal needle has particular characteristics. It is too fine for a stylet, and is therefore prone to blockage. CSF backflow is extremely slow, may not occur even when correctly placed, and it is hard to see down the metal hub required by fine needles. The high resistance to flow makes injection extremely slow, resulting in increased potential for poor mixing and inadequate spread. Our needles were 11 cm long and extended approximately 1 cm beyond the tip of a standard Tuohy needle. The hub was made in the shape of a male-to-female Luer fitting to stabilise during injection and this allowed the spinal needle to be fixed into the female Luer of the Tuohy.

In six of the 12 failed spinal anaesthetics, there was doubt that dural puncture had occurred; two of these were blamed on blocked needles. The remaining six failed because of inadequate spread in two, loss of local anaesthetic from the syringe during injection in two, and for the remaining two no reason was given. In the operating theatre we noticed a tendency for early regression of the block. We attributed this to slow injection speed and delay in placing the epidural catheter, which limited cephalad spread. The density of anaesthesia in the upper segments occasionally left something to be desired, while the height of the blocks was certainly satisfactory. A prophylactic epidural top-up was given after the birth of the baby because of this, with the patient lying flat.

The satisfactory spinal anaesthetics were a resounding clinical success in terms of efficacy and morbidity, but an overall 25% failure rate indicates that a 30-gauge spinal needle is probably not a practical proposition for routine clinical practice. It is possible that a 'through-the-Tuohy' technique may be prone to a higher failure rate because it is not CSF seeking, but this is only supposition at present. The optimal bore for obstetric spinal anaesthesia is not yet established. Significant possibilities for compromise exist between 26- and 29-gauge and further evaluation is warranted.

Acknowledgment

The authors thank Steriseal for providing the needles used in this study.

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Intrathecal anaesthesia for day-care surgery
A retrospective study of 160 cases using 25- and 26-gauge spinal needles

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Summary

The records of 160 day-care surgical patients who received intrathecal anaesthesia were reviewed. No major complications were recorded. The incidence of postspinal headache after puncture with a 25-gauge spinal needle was nearly four times more frequent compared to a 26-gauge needle. The occurrence of postspinal headache in patients over the age of 45 years was significantly less frequent ($p < 0.05$) than in younger patients. Intrathecal anaesthesia was acceptable to 91% of outpatients in this group.

Key words

*Anaesthesia, regional; intrathecal.
Surgery; day care.*

The benefits of day care surgery are now well established. Regional anaesthetic techniques offer several advantages to these patients: only the region that is being operated is blocked and residual analgesia is carried over to the post-operative period. The many side effects of general anaesthesia, such as nausea and vomiting, loss of coordination and residual hangover effect are avoided. Postanaesthetic nursing care is also easier.

There are only a few reports of intrathecal anaesthesia for day-care surgery,¹⁻⁴ but Atkinson and Lee⁵ wrote encouragingly about its feasibility. Extradural anaesthesia for day-care surgery is in common use in our hospital⁶ and intrathecal anaesthesia has also been used in 166 cases in the last year. We decided retrospectively to explore if intrathecal anaesthesia has any potential in this setting.

Methods

A questionnaire was sent within 7 days of surgery to 166 ASA 1 and 2 patients who had undergone surgery as day cases with the aid of intrathecal anaesthesia during the previous year. All operations performed are shown in Table 1 and their age distribution in Figure 1. Replies were received from 160 patients.

There were 92 males and 68 females; their ages ranged from 18 to 87 years. All patients were examined before the anaesthetic and deemed fit for day-care surgery. Consent was obtained for intrathecal anaesthesia and a careful

explanation of the anaesthetic method was undertaken by a member of the anaesthetic staff. No premedication was prescribed. All blocks were performed before 1200 hours.

A 25-gauge needle was used in 55 cases and a 26-gauge in the remainder. All blocks were performed without the aid of an introducer. The drug used in each case was heavy bupivacaine 0.5%. The patients received intravenous fluids, a vasopressor, sedatives and opioids during the operation as deemed necessary by the anaesthetist in charge.

The patients remained in the postoperative ward after the operation until they regained full motor and sensory function in their legs. All were tested for the return of perianal sensation and proprioception in their feet before ambulation was allowed. Patients were checked for any bladder distension and were catheterised if there was difficulty with micturition.

They were discharged home from the postoperative ward after a final check up by one of the anaesthetists. No special instructions about movement at home were given, but they were not encouraged to lie in bed for 24 hours. All patients were advised to contact the hospital in case of any unusual difficulties. No patient needed admission because of complications that resulted from intrathecal anaesthesia.

Any patient who started off as a day surgical case but required admission for surgical reasons because more than the intended operation had been performed, was not studied.

The Chi-squared test was used to evaluate statistical significance, using a p value of < 0.05 as significant.

Results

Eighty-four of the 160 patients experienced no problems whatsoever and stated that they would like to have the same anaesthetic again. Sixty-two patients experienced some problem or other during and after the operation, but nevertheless would prefer the same anaesthetic for a future procedure. Fourteen said that they would refuse a similar anaesthetic in future. The chief complaints are detailed in Table 2.

Postspinal headache occurred in 15 patients (9.4%, Table 3), but 10 of these stated that they would be prepared to have a similar anaesthetic in future. The other five

Table 1. Types of operations carried out on outpatients under intrathecal anaesthesia ($n = 160$).

Minor orthopaedic procedures (arthroscopy, arthroscopy + meniscectomy, hallux valgus correction, metatarsal osteotomies, Achilles tendon repair, removal of intramedullary nails)	55
Herniorrhaphy (femoral and inguinal)	52
Varicose vein surgery	28
Minor urological operations (orchidopexy, lithotripsy, hydrocoele operation, ureterolithotomy, urethrotomy)	16
Haemorrhoid operations	9

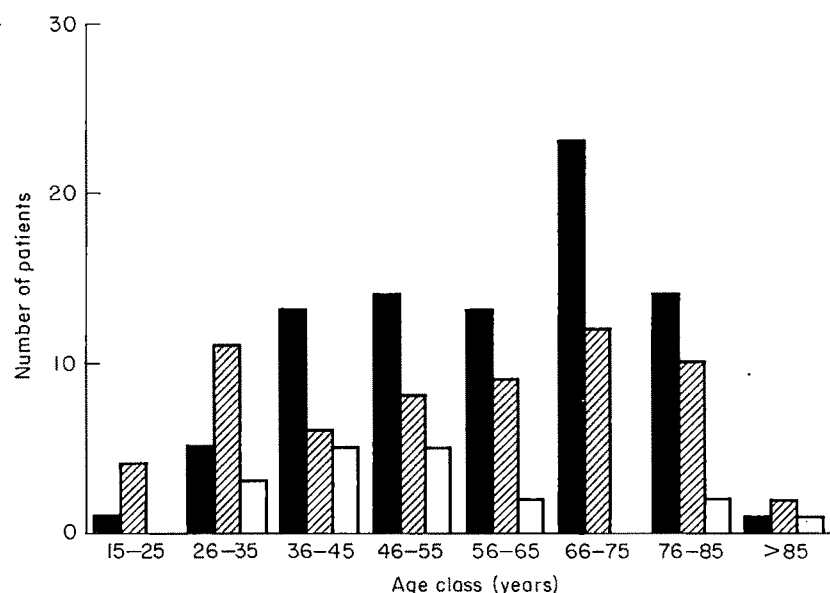


Fig. 1. Age distribution of patients ($n = 160$). ■, no problems ($n = 84$); these patients were entirely satisfied with the anaesthetic and experienced no postoperative problems as a result of the anaesthetic. ▨, positive group ($n = 62$); patients had one or more of the postspinal problems detailed in Table 2 yet preferred intrathecal anaesthesia for a future operation. □, negative group ($n = 14$); patients who would not have a similar anaesthetic again.

patients did not wish to have a spinal anaesthetic again. Two of these required a blood patch to cure their headache, and in both a 25-gauge needle was used. In all, a typical postspinal headache occurred in 10 of the 55 patients in whom a 25-gauge needle had been used (18.2%). This compared with an incidence of 4.8% (five out of 105 patients) after use of a 26-gauge needle. All these latter patients stated that they would have a similar anaesthetic again. Headache varied in duration from a few hours to 5 days. Headache incidence was not weighted towards one gender, but it was more frequent in patients under 45 years of age (Table 3).

Nearly 40 patients (25%) were catheterised in the postoperative period to relieve urinary retention. All patients could, however, pass urine without assistance within 8 hours of the spinal anaesthetic. However, 12 patients (7.5%) noted in the questionnaire that they experienced difficulty with micturition after discharge from the hospital. The problem lasted from a few hours to 5 days in one man. Patients undergoing herniorrhaphy had a higher frequency of micturition problems compared to other operations. This problem was commoner in men.

Twenty-six patients (16.25%) suffered backache of

varying severity, which lasted from 24 hours to up to a week. Treatment with mild analgesics resolved this problem in all cases. Twenty-six patients (16.25%) also experienced pain during the lumbar puncture itself, but the skin was not infiltrated with local anaesthetic before the puncture and none thought that this was a major drawback of this form of anaesthesia. Three patients (1.87%) had an unsatisfactory block and needed a general anaesthetic to complete the operation. All three patients said that they did not wish to have intrathecal anaesthesia in future.

Discussion

Intrathecal anaesthesia for day-care surgery is practised routinely in some centres in the USA^{1,2} and lumbar puncture for diagnostic purposes is also frequently carried out as day-stay procedures by neurologists.⁷ However, for day-care surgery it is still not widely accepted, mainly for fear of postspinal headache.

Atkinson and Lee⁸ blamed conservatism in British institutions as the cause for the resistance to spinal anaesthesia for day-care surgery. It appears that other countries also exhibit this conservatism if one is to explain the paucity of

Table 2. Side effects of intrathecal anaesthesia ($n = 160$).

	Problem free	Positive	Negative	Total
Back pain	0	18	8	26
Giddiness	0	15	4	19
Nausea	0	9	3	12
Vomiting	0	5	1	6
Postspinal headache	0	10	5	15
Difficulty in micturition	0	10	2	12
Pain during puncture	0	21	5	26
Unsatisfactory anaesthesia	0	0	3	3
Total number of patients	84	62	14	160

Problem free, patients who did not experience any problem during or after intrathecal anaesthesia. Positive, patients who had some problems as a result of the intrathecal procedure yet prefer a similar anaesthetic in future. Negative, patients who do not wish to have intrathecal anaesthesia in future.

Table 3. Distribution of postspinal headache according to age, sex and spinal needle size.

Age	Males	Females	Total	% of group
15-25	0	2	2	20.0
26-35	3	1	4	21.1
36-45	0	3*	3	13.0
46-55†	3*	3	6	19.3
56-65	2	0	2	8.7
66-75	0	0	0	0
76-85	0	0	0	0
>85	0	0	0	0
Total	8	7	15	9.37

*One patient in group received a blood patch for relief of postspinal headache.

†>45 years; $p < 0.05$.

publications on the subject. The incidence of postspinal headache is directly related to the bore of the needle used to puncture the dura and varies from 0.3% with a 26-gauge needle⁸ to up to 70% with an 18-gauge epidural needle.⁹ The incidence can be further reduced to 0% by using a 29-gauge needle.¹⁰

The frequency of postspinal headache decreases with age. Kortum *et al.*,¹¹ in a study that involved 3056 patients, reported a 33% incidence of headache in patients aged 20-29 years and 6.2% in patients aged 60-69 years using a 22-gauge needle. In our patients, only two (2.3%) in a group of 85 patients over the age of 55 years suffered a mild headache after intrathecal puncture. We did not notice any relationship between postspinal headache and the sex of the patient (8.7% for men and 10.3% for women) though it is believed that women suffer postspinal headache more often than men.¹¹

Efforts to reduce the incidence of postspinal headache by the use of prolonged bed rest^{12,13} and posture¹⁴ proved to be fruitless. It is possible to reduce the incidence further in younger ambulant people by using 29-gauge needles.¹⁰ Alternatively, epidural anaesthesia can be used for younger patients.⁶

Backache is a common problem after all types of anaesthesia¹⁵ and the incidence after spinal anaesthesia was reported to be between 2 and 25%.¹⁶ The exact aetiology is unknown in most cases. Various factors such as relaxation of the back musculature, trauma to the ligaments at the site of puncture, and a possible psychological overlay that arises from the fact that a lumbar procedure was performed, were suggested.¹⁶

Difficulty with micturition and retention of urine are common after many operations regardless of the type of anaesthesia used. The incidence of bladder dysfunction after gynaecological and obstetric procedures is high and in the region of 30%,¹⁷ but the incidence is not appreciably affected by using spinal anaesthesia.¹⁸ Axelsson *et al.*¹⁹ postulated that after spinal anaesthesia, sacral autonomic fibres are among the last to recover, and detrusor muscle contractions return very late, and believed that this may be the reason for the difficulty with micturition. Most of our patients received on an average 2.2 litres of crystalloid solutions and this would have contributed to distension of the bladder. Moreover, most people find it difficult to micturate while lying in bed.

Pflug *et al.*²⁰ have established criteria for ambulation after intrathecal anaesthesia. It is considered safe and orthostatic hypotension not a problem once there is full return of peri-anal sensation and proprioception in the feet.²⁰ Most of our patients fulfilled Pflug's criteria within 5.3 hours (range 3.0-8.0).

Intrathecal anaesthesia is easier to perform, quicker in onset and cost effective compared to epidural anaesthesia. Morbidity associated with its use can be minimised by proper selection of patients and use of finer needles. The incidence of postspinal headache is certainly more with intrathecal anaesthesia than with the epidural technique and anaesthesia cannot be prolonged beyond a certain time limit.

It was concluded that intrathecal anaesthesia for day-care surgery, with a 26-gauge needle, is suitable for patients above 55 years of age since they seem to be less susceptible to the development of postspinal headache. The 25-gauge needles are not recommended for day-care blocks.

Acknowledgments

We thank Dr J. Israelsson for his valuable comments and E. Bergh for her secretarial help. We are also grateful for the cooperation of all the nurses in the postoperative ward at Piteå General Hospital.

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Relief of injection pain in adults EMLA cream for 5 minutes before venepuncture

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Summary

The effectiveness of skin anaesthesia after 5 minutes' topical application of a lignocaine-prilocaine cream was evaluated. One hundred and twenty patients estimated the pain of antecubital venepuncture both on a linear scale and verbally after use of the cream for either 5 or 60 minutes, a placebo cream or no treatment. Reported pain was significantly less after only 5 minutes of the lignocaine-prilocaine cream ($p = 0.002$). The cream can be used to relieve the pain of all routine injections.

Key words

Anaesthetics, local; eutectic mixtures.

Needle puncture may be painful, is often unpleasant and occasionally can cause considerable distress in both children and adults. Many people regard the prospect of injections with great anxiety.¹ Dread of painful injection was admitted by 11 out of a 100 patients in one report.² Nevertheless, a recent survey of anaesthetic practice found that intravenous induction was used in 87.9% of cases undergoing general anaesthesia.³ Lignocaine and prilocaine have the property that crystals of the pure drugs combine to form an oily liquid at room temperature. EMLA (Astra Pharmaceuticals Ltd, Kings Langley), the acronym for 'eutectic mixture of local anaesthetics', contains lignocaine 25 mg/ml and prilocaine 25 mg/ml in a water-miscible base that is able to penetrate intact skin without loss of concentration.⁴ Previous studies have shown that painless injection is possible after EMLA, but application of the cream for 60 minutes is recommended.⁵⁻⁸ Would a shorter application time be effective clinically?

Patients and methods

The protocol was approved by the hospital ethics committee. One hundred and twenty patients due for morning blood tests took part in the study. They were asked only if they would consent to be included in a survey of injections. All were well, ASA status 1 or 2, and had taken no analgesics within the previous 4 hours. They were allocated systematically by date-of-birth to four treatment groups: lignocaine-prilocaine cream (EMLA), approximately 1.5 ml applied to either convenient antecubital fossa under an occlusive dressing for 60 minutes (E60); lignocaine-prilocaine, about 0.2 ml applied to the same site, rubbed in over a 5-cm wide area and the elbow flexed for exactly 5 minutes (E5); placebo, about 0.2 ml aqueous cream B.P. (emulsifying ointment 30%, chlorocresol 0.1%) from a similar tube applied in the same way for 5 minutes (PLAC5); and no treatment (NIL). There were equal numbers of men and women in each group. Needles of sizes 18, 20 and 22 standard wire gauge from a single manufacturer (Becton Dickinson UK Ltd, Cowley) were used in equal numbers in each group. Venepuncture was performed in the same way in every case by one of the authors (M.N.), who took care to allow the alcohol in spirit used for skin cleansing to evaporate to complete dryness.

Comments about anaesthesia and pain were avoided. Patients were asked by the same clinician, after the samples had been taken, to assess needle puncture pain on a 10-cm line marked 'no pain' (0) at one end and 'severe pain' (10) at the other. Assessments of pain on the linear scale were measured to the nearest mm. Particular care was taken to use neutral voice and gesture and a nonleading manner. They were also asked to estimate any pain as 'none', 'slight', 'moderate' or 'severe'. An impartial clinical assessment was made before the patients' evaluation. All patients were asked whether they were naturally left or right-handed. The results were examined by analysis of variance of the transformed linear scores (logarithm of (score + 1)), and by a less detailed examination of the verbal ratings.

Results

There were 60 males aged 18-86 (mean 66) years and 60 females aged 19-94 (mean 64) years. Nineteen patients (16%) were left-handed. The patients' assessments showed a skewed distribution: 13 scored zero. The clinician's assessments had a similarly skewed distribution: half the patients (60) scored zero. The two assessments were combined to give a better estimate of the (unknown) pain experienced. The frequency distribution of the combined scores, used for the main analysis, is shown in Figure 1. An analysis of the patients' assessments alone was performed separately.

Treatment group and needle size. Median values of the combined scores by treatment and needle group are given in Table 1; subgroups are relatively small, so minor differences should not be overemphasised. The patterns which emerge show that pain decreased from no previous treatment (NIL), with the highest pain score, through placebo (PLAC5), to the lignocaine-prilocaine cream applied for 5 minutes (E5) and 60 minutes (E60). Needle puncture after lignocaine-prilocaine cream applied for 5 minutes was significantly less painful than either placebo ($p < 0.01$) or no treatment ($p = 0.002$). The apparent response to placebo only was not significantly different from no treatment. No interaction was found between treatment and needles; the cream was effective whether the needles were large or small (Table 2). Patients who had the largest needles, 18 gauge, reported the most pain while those who

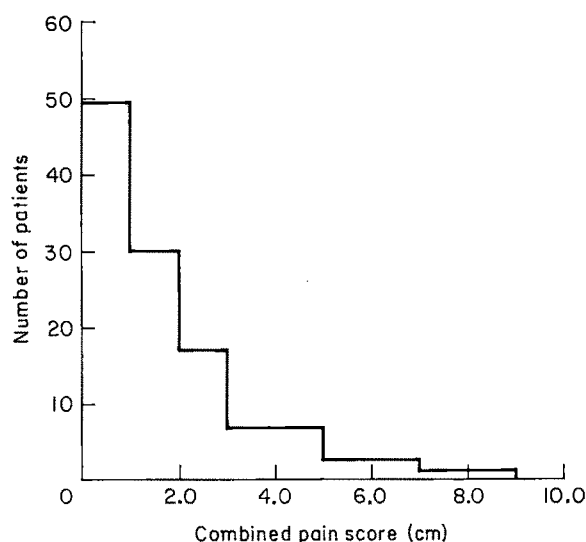


Fig. 1. Frequency distribution of the combined pain scores in all patients.

had the smallest, 22 gauge, reported the least ($p = 0.0001$). The analysis was repeated using the patients' responses alone and the patterns were found to be the same. These findings were confirmed by the verbal ratings. Many patients reported no pain while none admitted or were judged to have experienced severe distress.

Age affected reported pain. The youngest and oldest patients reported the most pain while those in between reported the least, and this effect is significant ($p \leq 0.02$). Chi-squared tests show that the observed pattern was not due to bias in the allocation of treatment or needle size between groups. The male patients reported less pain than the female patients, medians 0.9 and 1.3 respectively; the difference is significant ($p = 0.02$). A similar difference was the finding that 52% of male patients stated that they felt no pain compared with 40% of female patients. Interestingly, the sexes were rated almost equal by clinical assessment. Seventy per cent of the male and 73% of the female patients had 'no' reaction.

The left-handed patients reported less pain than the right-handed, and less pain was reported by the 57 patients in whom the nondominant side was used than by the 63 patients in whom venepuncture was carried out on the dominant side. These differences are not clinically significant.

Discussion

It was shown in young children that application of lignocaine-prilocaine cream for 30 minutes may be sufficient to prevent pain on cannulation, although in that study,⁹ it was compared with 45 minutes in adults. We set out to demonstrate the efficacy, if any, of application for 5 minutes. Our study was not blind, but use of the same observer was intended to ensure reliable comparisons. The intermediate position of results from the group where the cream was applied for 5 minutes, between 60 minutes and no active treatment, and the clear effect of needle size, suggest that this approach was valid. Assessments of the clinician were usually less than the patients' own estimations of their pain, with considerable individual variation. Our findings demonstrate that lignocaine-prilocaine cream applied for 5 minutes was effective. In addition, we found that fewer women than men reported no pain, with a characteristic difference revealed by occasional remarks. Male patients said that the needle was only just felt, so did not hurt, while

Table 1. Median combined pain scores, linear scale.

Treatment	Needle			
	22 g	20 g	18 g	All
E60	0.3	0.2	1.2	0.4
E5	0.4	0.5	1.1	1.0
PLAC5	0.8	1.4	2.3	1.9
NIL	1.6	1.2	2.8	2.3
All	0.5	0.9	1.9	

Table 2. Analysis of variance, main effects.

	Degrees of freedom	Sum of squares	F-ratio	p value
Needle	2	18.2946	9.89	0.0001
Treatment	3	24.1734	8.71	0.0001
Interaction	6	3.8222	0.69	0.6592
Residual	108	99.9031	—	—
Total	119	146.1933	—	—

female patients said it hurt slightly. Possible bias caused by the male gender of the clinician could not be ruled out.

The advantages of avoidance of painful needle puncture are self evident; not only is subjective discomfort reduced, but the procedure is also easier to carry out. Topical lignocaine-prilocaine cream may be a useful alternative to local anaesthetic infiltration where the latter would be painful or might obliterate anatomical landmarks.¹⁰ We have shown that application of EMLA cream for 5 minutes reduced significantly the amount of pain a patient was likely to experience during venepuncture. EMLA, as part of the procedure, also does much to relieve the anxiety about injections felt by so many patients. It should no longer be thought of as impractical because of a lengthy time of onset.

Acknowledgment

We thank Dr J.R.R. Stott for advice on the presentation of this report.

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Anaesthesia, 1990, Volume 45, pages 774-776

Successful difficult intubation Tracheal tube placement over a gum-elastic bougie

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Summary

A randomised study was carried out to assess the effect of tracheal tube rotation on the passage of a tube over a gum-elastic bougie into the trachea in 100 patients. The effect of the presence or absence of a laryngoscope on successful tube placement was also assessed. A grade 3 difficult intubation was simulated in patients with a laryngoscope. There was a significant difference in the rates for successful first-time intubation in those patients with tube orientation of -90° (with the bevel facing posteriorly) as compared with a tube orientation of 0° (the normal orientation with the bevel facing left). The unsuccessful first-time intubations with a 0° orientation were frequently converted to successful intubations with the -90° position at a second attempt. The presence of a laryngoscope in the mouth while rail-roading a tube over the bougie also made a significant difference to the rate of successful first-time intubations. The most successful method was to leave the laryngoscope in the mouth and rotate the tube to -90° .

Key words

Difficult tracheal intubation; gum-elastic bougie.

Successful management of a difficult intubation using a gum-elastic bougie is completed in two phases. The first involves passage of the bougie through the larynx into the trachea. The methods employed to recognise the position of the bougie in the trachea when the vocal cords are not visible were previously investigated in this centre.¹

The tracheal tube is passed over the bougie into the trachea in the second phase. The successful placement of a bougie through the larynx, in our clinical experience, may frequently be followed by difficulty in this second phase. Cossham² has suggested that rotation of the tracheal tube a quarter-turn anticlockwise may improve the success rate in these circumstances, although this has never been quantified. Therefore, a systematic study was conducted to investigate the effect of anticlockwise rotation of the tracheal tube over the bougie. We also assessed whether the presence or absence of a laryngoscope in the mouth influenced the passage of the tracheal tube because some anaesthetists withdraw the laryngoscope before passing the tube over the bougie.

Methods

Informed consent was obtained from all patients, after approval from the Ethics Committee. One hundred patients aged 18-70 years, in ASA grades 1 and 2, for whom tracheal intubation was planned as part of the anaesthetic sequence were studied. Patients were excluded from the study if they were to have ocular or neurosurgical procedures; those patients with asthma and in those cases when cricoid pressure was to be used were also excluded.

The normal orientation of the tracheal tube with the

bevel to the left will be referred to as the 0° (neutral) position; the orientation of the tube with a quarter-turn anticlockwise such that the bevel faces posteriorly will be referred to as the -90° position. The Portex standard cuffed tracheal tubes used were of 9 mm internal diameter for men and 8 mm for women.

The ECG, blood pressure and oxygen saturation (by pulse oximeter) were monitored in all patients throughout the period of anaesthesia. Patients were pre-oxygenated for 3 minutes. Anaesthesia was induced with an intravenous agent chosen by the anaesthetist present. Muscle relaxation was achieved by the administration of suxamethonium 1.5 mg/kg intravenously. Laryngoscopy was performed after 60 seconds using a Macintosh blade, size 3.

The initial view of the glottic structures was classified according to Cormack and Lehane:³ grade 1, the glottis can be fully exposed (including anterior and posterior commissures); grade 2, the glottis is partially exposed (posterior commissure only); grade 3, the glottis cannot be exposed (epiglottis visible); grade 4, neither the glottis nor the epiglottis can be exposed.

A 15-FG gum-elastic bougie (Eschmann, UK) was passed into the trachea after laryngoscopy. The patients were randomly allocated to one of four groups A, B, C and D (25 in each group). In groups A and B the laryngoscope was removed from the mouth after insertion of the bougie before attempted intubation. In group A the tube was at 0° for the first attempt; in group B at -90° (i.e. with a quarter-turn anticlockwise). If unsuccessful, the tube was pulled back 2 cm and rotated to -90° for group A and to 0° for group B for a second attempt. A grade 3 intubation was simulated in groups C and D as described by Cormack

Table 1. Success at intubation in the four groups.

Group	Laryngoscope	First attempt		Second attempt		Overall success	Combined rates
		Position	Success rate	Position	Success rate		
A	No	0°	2/25 (8%)	-90°	18/23 (78%)	20 (80%)	{30/50}
B	No	-90°	9/25 (36%)	0°	1/16 (6%)	10 (40%)	
C	Yes	0°	12/25 (48%)	-90°	10/13 (77%)	22 (88%)	{47/50}
D	Yes	-90°	25/25 (100%)	0°	—	25 (100%)	

and Lehane:³ with the laryngoscope in position the epiglottis was allowed to fall so as to obscure the cords. The first attempt in group C was with the tracheal tube at 0° and in group D at -90°. The tube was rotated to the alternate position for the second attempt, as described above, if the first attempt was unsuccessful. The study was abandoned if both attempts at intubation according to the protocol failed. Intubation was then performed in the usual way.

Results

There was a wide spectrum of results for the first attempt that varied from a success rate of only 8% in group A (no laryngoscope/0°) in group D (with laryngoscope/-90°) (Table 1). The overall success rates, after a second attempt where necessary, ranged from 40% in group B to 100% in group D. The combined success rate at the first attempt for the 0° position (with or without a laryngoscope) was 14/50 (28%); for the -90° position it was 34/50 (68%) ($p < 0.001$ by Fisher exact test). The combined success rate at the first attempt when no laryngoscope is used (in 0° or -90° position) was 11/50 (22%) whereas with a laryngoscope it was 37/50 (74%) ($p < 0.000001$). The overall success rate, after both tube positions had been tried where necessary, was 30/50 (60%) without a laryngoscope compared with 47/50 (94%) with a laryngoscope ($p < 0.0001$).

The initial views at laryngoscopy were grade 1 in 61 patients; grade 2 in 35 patients; grade 3 in four patients, and no patients in grade 4. There were thus four cases in this group of 100 patients in whom the bougie was required to facilitate intubation, three of whom were in group C and one in group D. All four grade 3 patients were successfully intubated. Two in group C were intubated at the first attempt and one at the second attempt i.e. with tube at -90°.

Discussion

The results clearly show the superiority of the -90° position as suggested by Cossham. First-time use of the -90° position with the retention of the laryngoscope in the mouth resulted in maximal success. A significant number of the first-attempt failures using the 0° position were successfully intubated at the second attempt by rotating the tube to the -90° position (groups A and C).

Intubation in the four genuine grade 3 views of the glottic structures was successfully achieved in all patients with the laryngoscope in the mouth. The one failure in group C with the tube position at 0° was converted to a successful intubation by changing the tube position to -90°. This one patient supports the use of -90° rotation of the tube, as is also shown in the simulated difficult intubations in group C.

All the patients in this study with tube position at -90° and with the laryngoscope in the mouth were successfully intubated. However, failure might occur and in these cases

it might be worthwhile rotating the tube to 0° since this was successful in one case in group B (1/16 i.e. 6%).

A likely explanation for the better results with the -90° position emerges from the comments made by the investigators: there were 13 cases where the site of obstruction to the passage of the tube over the bougie was noted. The tube was observed to lie behind the glottis in the midline in eight of these cases. In three cases the tube went straight back and the tip stuck on the posterior pharyngeal wall. In one case the epiglottis obstructed the tip of the tube. There was one case where the tube was obstructed just after its passage through the vocal cords. On passage of the tube in the -90° position the bevel of the tube rides up the slope of the soft tissue behind the larynx and enters the trachea without hold-up. On no occasion was the tube obstructed by the right vocal cord as has been mentioned in the literature.^{2,3}

It was noted that the flexible gum-elastic bougie often curved anteriorly from the posterior pharyngeal wall. The presence of the laryngoscope allows the bougie to retain a straighter line and thus facilitate intubation. Further study with regard to the optimum size and flexibility of the bougie both for insertion into the trachea and later passage of the tube over the bougie is needed. The use of the gum-elastic bougie, where the view of the cords was obscured by the tracheal tube, was pioneered in 1949 by Macintosh.⁴

There is considerable uncertainty about the incidence of difficult intubation in the population. Estimates vary: 3.6%,⁵ 2.3%⁵ and 1% (Cardiff Anaesthetic Record System 1972-79) have been reported, but in none of these was there any systematic classification of glottic views. This paper shows an incidence of 4% of grade 3 glottic views, but with a sample size of only 100 patients the 95% confidence interval for a true incidence extends from 1% to 10%.⁶

Mismanagement of difficult intubation is a significant cause of morbidity and mortality under anaesthesia.⁷⁻⁹ Severe difficulty may sometimes be anticipated by thorough clinical examination and the presence of features such as a short, thick neck, receding jaw, the view of the oropharyngeal structures and prominent teeth.^{10,11} Adequate preparation in such severe cases may allow the use of sophisticated approaches such as fiberoptic or retrograde intubation. However, difficult cases are often not predictable^{7,12} and many clinicians will not have been trained in the complex methods or may not achieve the level of competence which may be required for use in a difficult and urgent intubation. Furthermore, sophisticated equipment may not be readily available in all anaesthetic locations and this further underlines the need for correct use of a simple and reliable technique.

One of the simple aids to unexpected difficult intubation is the gum-elastic bougie. We recommend that all trainees at an early stage in their career should be taught how to use the gum-elastic bougie during a simulated difficult intubation in association with an optimum technique for correct

tracheal tube placement over the bougie. The technique of rotating the tube to -90° should be emphasised. It is also important to note that holding the bougie near its distal angulated end with a pair of Magill forceps will enable forward movement of the tip of the bougie to be achieved, and will more effectively facilitate passage of the bougie when difficulty occurs. Regular practice and familiarity with this drill should help in the management of a truly difficult intubation.

We conclude that in cases of unexpected difficult intubation first-time successful placement of the tracheal tube over the gum-elastic bougie will be greatly increased by the presence of a laryngoscope in the mouth and by rotating the tube to -90° anticlockwise thus keeping the bevel of the tube posterior. This will also decrease trauma and the incidence of protracted attempts at intubation and reduce the need for more sophisticated techniques. The incidence of failed intubation should also be reduced.

Acknowledgments

The authors are extremely grateful to Professor W.W. Mapleson for his valuable assistance and advice in the preparation of this paper.

Anaesthesia, 1990, Volume 45, pages 776–780

Acid aspiration prophylaxis in 288 obstetric anaesthetic departments in the United Kingdom

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Summary

The choice of drugs for acid aspiration prophylaxis in 288 obstetric anaesthetic departments in the United Kingdom was determined by questionnaire. The results are compared with a similar survey of anaesthetic departments 5 years ago. The prescription of sodium citrate and the H_2 receptor antagonist ranitidine has superseded that of Mist. magnesium trisilicate.

Key words

Anaesthesia; obstetric.

Complications; aspiration, prophylaxis.

Acid aspiration syndrome (AAS) or Mendelson's syndrome¹ continues to be a significant cause of maternal mortality after Caesarean section in the United Kingdom.^{2,3} Taylor and Pryse-Davies showed in 1966 that neutralisation of gastric fluid with magnesium trisilicate mixture prevented the severe pulmonary complications of acid aspiration,⁴ and it has been recommended that all women in active labour and before induction of anaesthesia should be starved and receive acid aspiration prophylaxis.⁵ This has now become standard practice in most departments throughout the United Kingdom.

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In 1984 a survey of 291 anaesthetic departments showed that the most popular acid aspiration prophylaxis used was Mist. magnesium trisilicate.⁶ A considerable body of experimental evidence has been published, since the introduction of the H_2 receptor antagonists cimetidine and ranitidine, that demonstrate their effectiveness at reducing gastric acidity.^{7–13} It was decided, in view of the efficacy of these newer agents and their apparent increasing popularity, to carry out a further survey to assess whether acid aspiration prophylaxis prescribing had changed over the past 5 years in the United Kingdom.

Questionnaire

001683

Office Use Only
(7) (8) (9)

1. Is obstetric anaesthesia carried out in your department?

Please tick (✓) appropriate box:

Yes ☐ No ☐ (10)

2. If yes, approximately how many deliveries per year?

Please tick (✓) appropriate box:

Less than 500	<input type="checkbox"/> 1	2000-3000	<input type="checkbox"/> 4	(11)
500-1000	<input type="checkbox"/> 2	3000-4000	<input type="checkbox"/> 5	
1000-2000	<input type="checkbox"/> 3	More than 4000	<input type="checkbox"/> 6	

3. Is chemoprophylaxis against acid aspiration syndrome normally given to patients who are in *active labour* in your department?

Please tick (✓) appropriate box:

Yes ☐ No ☐ (12)

Which of the following agents are normally used, how frequently and by what route are they given (O, IM, IV)?

Please tick appropriate

	box	Route	Amount	Frequency	
Magnesium trisilicate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(13)
0.3 M Sodium citrate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(14)
Cimetidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(15)
Ranitidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(16)
Others (please specify)	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(17)

(18)
(19)
(20)
(21)

4. For patients undergoing ELECTIVE Caesarean section is chemoprophylaxis against acid aspiration syndrome normally carried out in your department?

Please tick (✓) appropriate box:

Yes ☐ No ☐ (22)

Which of the following agents are normally used, how frequently and by what route are they given (O, IM, IV)?

Please tick appropriate

	box	Route	Amount	Timing	
Atropine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(23)
Metoclopramide	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(24)
Magnesium trisilicate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(25)
0.3 M Sodium citrate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(26)
Cimetidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(27)
Ranitidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(28)
Others (please specify)	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(29)

(30)
(31)
(32)
(33)

5. For patients undergoing EMERGENCY Caesarean section is chemoprophylaxis against acid aspiration syndrome normally carried out in your department?

Please tick (✓) appropriate box:

Yes ☐ No ☐ (34)

Which of the following agents are normally used? How much is given, by what route (O, IM, IV) and what is the timing in relation to induction of anaesthesia?

Please tick appropriate

	box	Route	Amount	Timing	
Atropine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(35)
Metoclopramide	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(36)
Magnesium trisilicate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(37)
0.3 M Sodium citrate	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(38)
Cimetidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(39)
Ranitidine	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(40)
Others (please specify)	<input type="checkbox"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	(41)

(42)
(43)
(44)
(45)

6. Additional comments if appropriate:

(46)
(47)
(48)
(49)
(50)

Fig. 1. The questionnaire used in the survey.

Method

A questionnaire was designed and sent to consultant anaesthetists in the United Kingdom (Fig. 1). Respondents were initially asked whether obstetric anaesthesia was carried out in their department, and if so how many deliveries were managed in the hospital each year. The questionnaire was then divided into three sections and dealt with acid aspiration prophylaxis during normal labour, for elective Caesarean section, and for emergency Caesarean section.

A list of commonly used drugs was given in each section. The respondent was asked to tick the appropriate agent and give details about dosage, route, frequency and timing of administration of the drug in relation to the induction of anaesthesia. Finally, respondents were asked to append any other details they considered relevant. The questionnaire was sent out in November 1988 using the mailing list of a large pharmaceutical company. Each questionnaire was coded to avoid duplication and to facilitate in the analysis of the data. A stamped addressed envelope was enclosed.

Results

Questionnaires were sent to 299 departments, and 288 completed replies were received: a 96.3% response rate. The results of the various regimens used by the departments may be considered under three headings.

Active labour (Table 1). No prophylaxis of any kind was used in 73 of 288 departments which represents 25% of the total. Of the 214 departments where regular prophylaxis was administered, 172 (80%) used the H₂ receptor antagonist ranitidine. This was used as the sole agent in 104 departments (48%), and combined with sodium citrate in 57 departments (27%). Magnesium trisilicate was used in only 31 departments (14%).

Elective Caesarean section (Table 2). In this group 284 departments (99%) used some form of acid aspiration prophylaxis. Sodium citrate was used either alone or in combination by 222 departments (78%), while the ranitidine/sodium citrate combination was used by 199 of the

Table 1. Acid aspiration prophylaxis for patients in active labour.

	Yes	No	Oral	Intramuscular injection	Intravenous injection
Total (288 departments)	215 (75%)	73 (25%)			
Ranitidine	172 (80%)	—	53 (89%)	12 (7%)	7 (4%)
Sodium citrate	76 (35%)	—	76 (100%)	—	—
Magnesium trisilicate	31 (14%)	—	31 (100%)	—	—
Cimetidine	9 (4%)	—	9 (100%)	—	—
Ranitidine only	104 (48%)				
Ranitidine and sodium citrate only	49 (23%)				
Ranitidine and sodium citrate and others	8 (4%)				
Ranitidine and other drugs (not sodium citrate)	11 (5%)				

Table 2. Acid aspiration prophylaxis for elective Caesarean section.

	Yes	No	Oral	Intramuscular injection	Intravenous injection
Total (288 departments)	284 (99%)	4 (1%)			
Ranitidine	247 (87%)	—	224 (91%)	18 (7%)	5 (5%)
Sodium citrate	222 (78%)	—	222 (100%)	—	—
Magnesium trisilicate	23 (8%)	—	23 (100%)	—	—
Metaclopramide	96 (34%)	—	34 (35%)	44 (45%)	8 (20%)
Atropine	15 (5%)	—	—	6 (40%)	9 (60%)
Cimetidine	14 (5%)	—	12 (86%)	2 (14%)	—
Glycopyrronium	3 (1%)	—	—	1 (33%)	2 (67%)
Sodium bicarbonate	3 (1%)	—	3 (100%)	—	—
Ranitidine only	28 (10%)				
Ranitidine and sodium citrate only	110 (39%)				
Ranitidine, sodium citrate and others	89 (31%)				
Ranitidine and other drugs (not sodium citrate)	20 (7%)				

Table 3. Acid aspiration prophylaxis for emergency Caesarean section.

	Yes	No	Oral	Intramuscular injection	Intravenous injection
Total (288 departments)	288	0			
Ranitidine	178 (62%)	—	55 (31%)	27 (15%)	96 (54%)
Sodium citrate	247 (86%)	—	247 (100%)	—	—
Magnesium trisilicate	29 (10%)	—	29 (100%)	—	—
Metaclopramide	102 (35%)	—	—	46 (46%)	56 (54%)
Atropine	18 (6%)	—	1 (6%)	3 (17%)	15 (83%)
Cimetidine	19 (7%)	—	2 (10%)	11 (58%)	6 (32%)
Glycopyrronium	2 (1%)	—	—	—	2 (100%)
Sodium bicarbonate	5 (2%)	—	—	—	—
Ranitidine only	2 (1%)				
Ranitidine and sodium citrate only	87 (30%)				
Ranitidine, sodium citrate and others	74 (26%)				
Ranitidine and other drugs (not sodium citrate)	15 (5%)				

284 departments (69%). Magnesium trisilicate was used in only 23 departments (8%) and cimetidine in 14 departments (5%). Atropine was used by 15 departments (5%), while 96 departments used metoclopramide routinely (34%).

Emergency Caesarean section (Table 3). A similar pattern of prescribing emerged in this group, where 100% of departments used some form of acid aspiration prophylaxis. Again the ranitidine/sodium citrate combination was by far the most popular; it was used by 161 (56%) departments, and magnesium trisilicate was used routinely in only 29 (10%) departments. Atropine was used by 18 departments (6%), while metoclopramide was used in 102 departments (35%).

The distribution of the number of deliveries per year in each department is shown in Table 4.

Table 4. The number of deliveries per year in each department.

0–500	4	(1.5%)
500–1000	12	(4%)
1000–2000	62	(21.5%)
2000–3000	97	(34%)
3000–4000	57	(19.5%)
4000 and over	56	(19.5%)
Total	288	

Discussion

The 288 replies received in this survey are similar in number to the 291 replies analysed 5 years ago,⁶ and allows a reasonable comparison between the two surveys.

At present 25% of departments give no acid aspiration

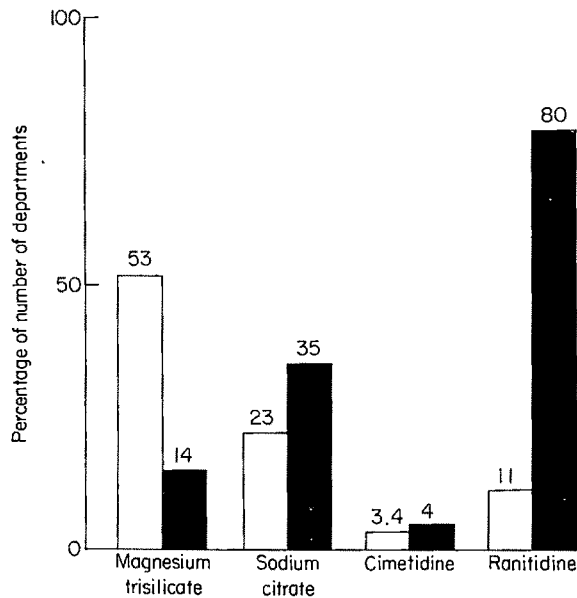


Fig. 2. Acid aspiration prophylaxis for patients in active labour. A comparison of two surveys carried out in March 1984 (light bars) and November 1988 (dark bars).

prophylaxis to women in active labour compared with 18% of departments 5 years ago. This may not be a true reflection of current practice since many departments stated that while prophylaxis was not routinely given to all labouring mothers, it was given to those high-risk patients more likely to require instrumental delivery or Caesarean section. It is interesting to speculate why a further 7% of departments do not use regular prophylaxis during active labour. They may have reservations about the rationality of current prescribing and doubts as to the efficacy of such treatment, or a belief that it is simply impractical to carry it out. There may be an increase in the view that the cost or the discomfort caused to the patient is simply not justified by any theoretical benefit which, even if possible to quantify, would probably be extremely small.¹²

Eighty per cent of the departments that administer regular prophylaxis in labour now use ranitidine, in contrast to 5 years ago when 54% of departments used magnesium trisilicate, and only 11% used ranitidine (Fig. 2). The number of departments in the elective Caesarean

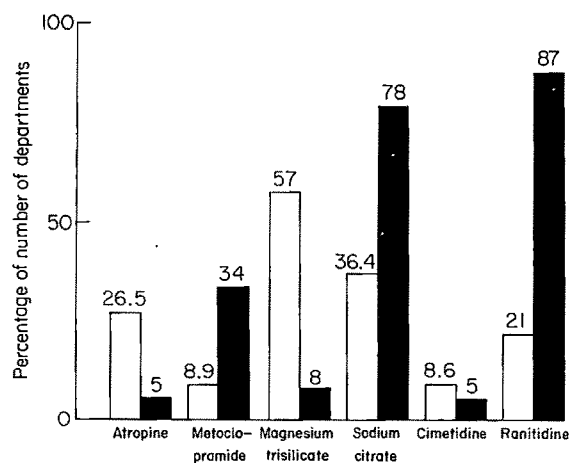


Fig. 3. Acid aspiration prophylaxis given to patients who have elective Caesarean section. A comparison of two surveys carried out in March 1984 (light bars) and November 1988 (dark bars).

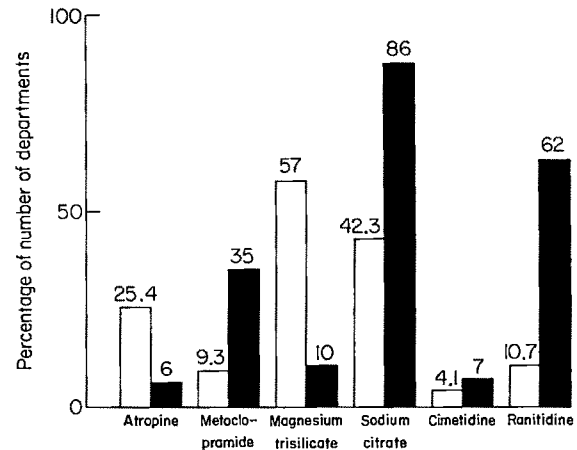


Fig. 4. Acid aspiration prophylaxis given to patients who have emergency Caesarean section. A comparison of two surveys carried out in March 1984 (light bars) and November 1988 (dark bars).

section group that give acid aspiration prophylaxis are comparable; 99% now do so compared to 98.6% in the previous survey. There is increased popularity of the ranitidine/sodium citrate combination, whereas there has been a decrease in the use of magnesium trisilicate from 57% to only 8% of departments at present. The use of atropine has declined while that of metoclopramide has increased (Fig. 3). All departments in both surveys used some form of acid aspiration prophylaxis for emergency Caesarean section. The trend is towards the use of the ranitidine/sodium citrate combination; only 10.7% of departments used it 5 years ago, while 57% of departments used magnesium trisilicate (Fig. 4). The increasing popularity of metoclopramide in preference to atropine, is probably because of its ability to increase the rate of gastric emptying in labouring women,¹⁴ whereas atropine reduces the tone of the lower oesophageal sphincter.¹⁵

Several reasons may be responsible for the swing away from magnesium trisilicate. Reports of fatal or near fatal sequelae in patients who have aspirated despite having been given magnesium trisilicate may have contributed to this loss of confidence.^{2,16} Magnesium trisilicate, in contrast to sodium citrate, mixes poorly with gastric contents,¹⁷ while its particulate nature causes concern because other particulate antacids give rise to pneumonitis.¹⁸ The 2-hourly administration regimen recommended by Crawford is inconvenient and may leave 20% of patients unprotected during the subsequent induction of anaesthesia,¹⁹ while even in highly motivated units there is a failure to follow the suggested protocol in 18% of cases.²⁰ These factors, coupled with published studies from several centres that show the efficacy of the H₂ receptor antagonists at reducing gastric acidity,⁷⁻¹² have probably all contributed to this swing away from magnesium trisilicate. The combination of ranitidine and sodium citrate in particular has received much attention and would appear to be the yardstick against which other regimens should be measured.¹³ Ranitidine is the preferred H₂ receptor antagonist; it is readily absorbed orally and has an 8-hour duration of action. It does not inhibit drug-metabolising enzymes,²¹ in contrast to cimetidine, and fears that it may have adverse effects on the unborn baby or neonate have not been confirmed. Ranitidine given intravenously was recently reported to cause cardiac arrest, but this was not in an obstetric patient.²²

The results of the survey reflect the overall opinion that acid aspiration prophylaxis is necessary before elective and

emergency Caesarean section, and the regimen of choice at present is the ranitidine and sodium citrate combination. There is controversy about giving prophylaxis to all women in active labour, but oral ranitidine given 6 hourly would appear to be the drug of choice.

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Anaesthesia and the law

The series *Anaesthesia and the Law* by Mrs D. Brahams is quite interesting. The material she uses is in the public domain, and is derived from court transcripts and judgements, which are available at a cost to the public at large. Thus all the details of a case can be found, if one so desires.

However in the April issue of *Anaesthesia* in the article entitled 'Series of errors which culminated in death' both the name of the patient and the name of the anaesthetist concerned were quoted. The details of the case at the Coroner's Court are of interest as are the comments of the author, but I think it is wrong to mention a patient by name in a medical journal. It is, however, far worse and in my opinion quite unwarranted, to publish the name of a colleague who has also suffered the trauma of this tragedy in the house journal of the Association of Anaesthetists. There can be no good reason for publishing either name.

D. Brahams (*Anaesthesia* 1990; 45: 332–3) asks why a junior anaesthetist made a series of errors that culminated in the death of a patient but fails to point out the one obvious answer. The incident occurred in the early hours of 23 January 1989, which was a Monday, so we can probably assume that the anaesthetist had been on duty for the previous 39 hours. This case illustrates the urgent need to address the problem of long periods of continuous duty in a specialty that requires continual vigilance and alertness.

In addition, I was amazed to see the anaesthetist's name appear. He (she) suffered the trauma of an inquest and then has the case paraded before his (her) colleagues with no opportunity to defend or justify his (her) actions. Anonymity is necessary to prevent this instructive series of articles becoming a kangaroo court.

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A reply

We have received, at long last, a few letters which comment on the quotation of patients', anaesthetists', and hospitals' names in the series *Anaesthesia and the Law*. The letters came from Dr P. Cox (Sweden), A.P. Vickers (Lancaster), C.E. Blogg (Oxford), J.P. Curran (Nottingham), G. Morgan (Truro), J.M. McDowell (Keighley) and M. Laban with C. Heneghan (Ealing). Each letter makes similar points to those made by Dr T.H. Taylor (of the Medical Protection Society) and these include the observation that the individual anaesthetist has suffered enough and that publication of names in *Anaesthesia* is against honourable traditional practice. Furthermore, identification of the

location of the hospital in the most recent article (*Anaesthesia*, 1990; 45: 332-3) was detrimental to a large number of anaesthetists. Naturally, all the facts are already a matter of public record although few had reached the national press.

There is, I believe, a case to answer. Moreover in this most recent example I believe I was mistaken in leaving the names of the individuals in the manuscript. However, there are other facts and opinions which are relevant and are expanded below by Mrs Brahams.

J.N. LUNN
Editor

Accountability and anonymity

Anaesthesia decided to launch a new series of educational articles entitled *Anaesthesia and the Law* in January 1989. The Editor wrote in an editorial which accompanied the first report: 'It may be useful for our readers to be aware of the facts as they appear to lawyers without the embellishment of journalists or the delay caused by medical protection mechanisms . . . even in serious cases, in which negligence is admitted and for which no defence can be offered the settlement is made out of court and the defendant remains anonymous . . . The contrast between ourselves and another learned profession is even more remarkable. The Law Society's Gazette regularly publishes a list of the names of lawyers who have offended their professional code . . . Is it time for more openness in medicine? — then at least, our critics could no longer accuse us of protective self interest.'

Sixteen months after this and after the publication of a report entitled 'Series of errors which culminated in death' (*Anaesthesia* 1990; 45: 332-3) there is a response (hostile) to the decision to include the names of patients, anaesthetists and hospitals in *Anaesthesia and the Law*. There is no breach of confidentiality since all the information used is in the public domain, but anaesthetists' eyebrows and indeed hackles are raised, it seems, by their publication in a 'professional journal'. One particularly ferocious critic's view was that such information was more suited to 'the tabloid press'. But this information is also contained in the law reports of *The Times* and the *Independent*! Sad to say, the 'righteous' indignation prompted by the inclusion of the name of the patient in these reports seems to have been far outweighed by the outrage perpetrated by the naming of the doctor and the hospital concerned. Such outrage was clearly not experienced by those same readers when dentists and their patients were named in the preceding reports.

To put matters into a proper perspective, eyebrows would certainly be raised in other professions if in any article which described a factual incident involving a negligent professional practice or other professional shortcomings of practice, the parties concerned were referred to anonymously as 'a client', 'a solicitor', 'a barrister', 'a structural engineer', 'a surveyor'. This may be appropriate in an insurance and protection context (such as the annual reports published by the Medical Defence Union and the

Medical Protection Society) but surely not in an open journal published in an 'open' society?

One of the chief stated concerns of the Association for Victims of Medical Accidents is to bring about the greater accountability of doctors to their patients. Doctors have always regarded themselves as being at the top of the professional pile; but their claim to this superior ethical stance is hardly supported by their protective and defensive response which, in my view, does them little credit.

Having said that, the aim of these articles is to educate, and to alert doctors to potentially dangerous practices, equipment failures, staffing levels and systems of working. It is not to pillory or punish individual doctors and (or) the hospitals or the health authorities who run them, but to call them to account seems not inappropriate.

Readers may wish to bear in mind Rudyard Kipling's words:

'I have six honest serving men,
They taught me all I knew,
Their names are What and Why and When
And How and Where and Who.'

Some readers would like the last two serving men dispensed with, preferring anonymity to accountability. This is a cause for much criticism by the public in many other fields, particularly when government chooses to invoke the Official Secrets Act. By exerting pressure on us, could anaesthetists not fairly be accused of trying to create such an unwritten law of their own to the detriment of the public interest (which of course is not always synonymous with that which is of interest to the public and the profession). The purpose of law reports in the public domain is to inform; names of individuals are only there normally withheld if publication is unduly prejudicial, such as in the case of minors, rape victims, mental patients and those suffering from sexually transmitted diseases or who might otherwise be put at risk.

The editor has, however, noted the objections voiced by some readers and will continue to exercise his discretion in this matter in all future publications.

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D. BRAHAMS

Children's premedication and hypoxaemia

Premedication in children continues to be a subject for debate; some anaesthetists use sedative regimens to decrease distress and struggling at induction, whilst others prefer to use no premedicant at all. The potential for

hypoxaemia in children begins with the administration, if any, of the premedicant drugs.

Opioids are well known to cause respiratory depression, but children premedicated with trimeprazine, atropine and

Prescribing Information

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Warnings: **Contraindications** Concurrent MAOI administration, left ventricular outlet obstruction (such as hypertrophic obstructive cardiomyopathy or aortic stenosis), pheochromocytoma, thrombocytopenia. **Precautions** If any correction of hypovolaemia is required this should be achieved before the administration of Dopacard. **Warnings** Dopacard should be administered with caution to patients with acute myocardial infarction or recent episodes of angina pectoris. A fall in circulating platelet numbers has been observed in some patients. No adverse experiences attributable to alterations in platelet count have been seen in clinical studies. Plasma potassium may decrease and blood glucose may increase during Dopacard administration and care is required in its use in patients with, or at risk of, hypokalaemia or hyperglycaemia. There is no evidence to suggest that Dopacard has significant arrhythmogenic potential. However, if cardiac arrhythmia occurs during administration a reduction or temporary discontinuation of the infusion should be considered. The safety and efficacy of Dopacard for use in children has not been established.

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Dopacard is not currently recommended for use in pregnant and lactating women. **Side effects**

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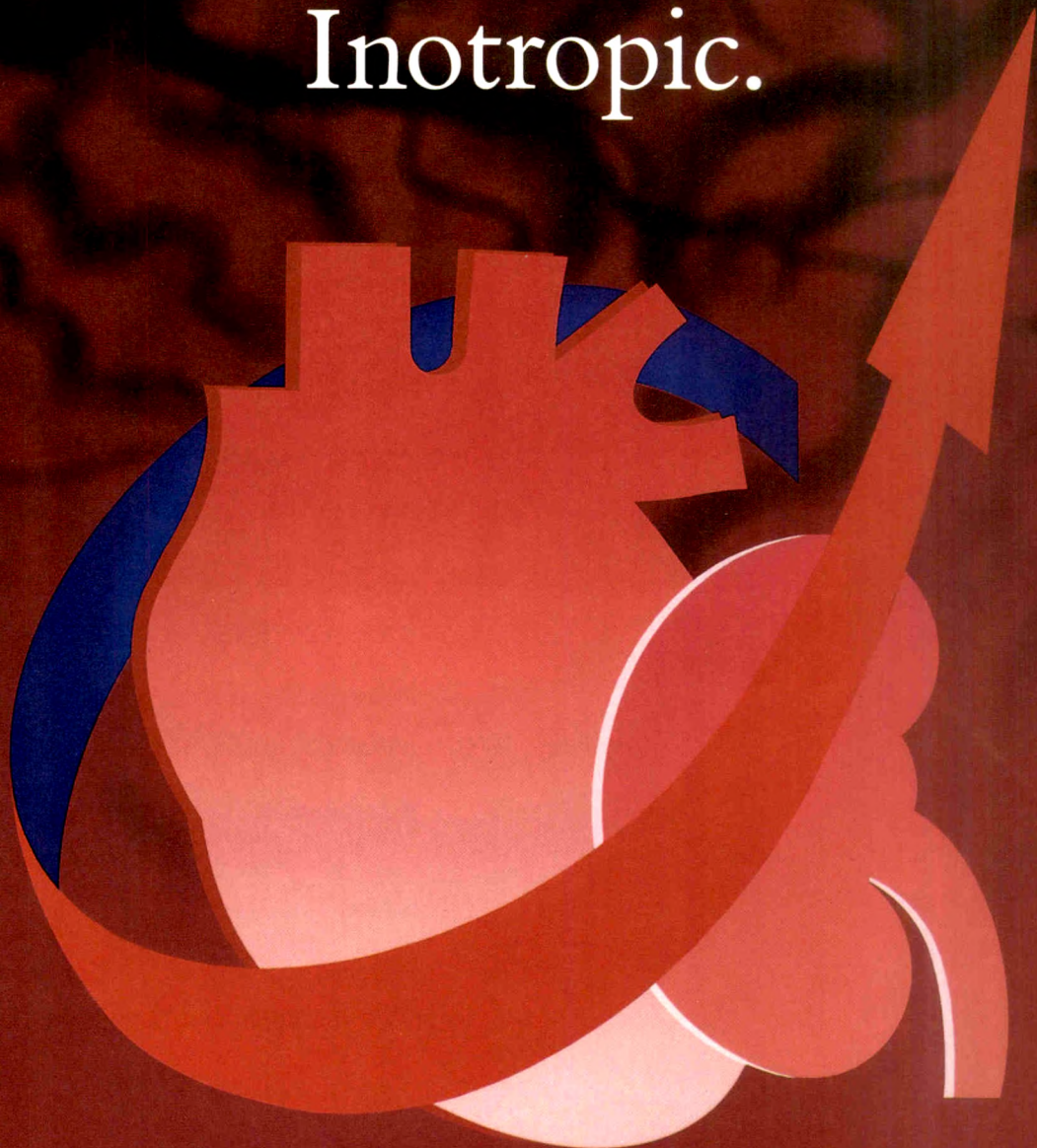
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morphine had the least incidence of reduction in oxygen saturation at induction.¹ However, since hypoxic events are evenly distributed throughout induction, maintenance and recovery,² it is desirable to know what influence opioids have after induction on oxygen saturation.

The incidence of hypoxaemia in the recovery room was compared after three sedative premedication regimens.

Ninety children aged from 2 to 8 years (inclusive) were divided randomly into 3 groups of 30. Group A received trimeprazine 2 mg/kg and atropine 0.02 mg/kg orally, both 2 hours before operation. Group B received pethidine 1 mg/kg and atropine 0.02 mg/kg intramuscularly 1 hour before operation. Group C received intramuscular morphine 0.2 mg/kg and atropine 0.02 mg/kg 1 hour before operation. All children underwent elective ENT, ophthalmic or general surgery and were graded ASA 1 or 2.

Anaesthesia was induced with thiopentone 4 mg/kg and tracheal intubation was achieved in all children after suxamethonium 1 mg/kg. Anaesthesia was maintained with spontaneous breathing of oxygen, nitrous oxide and halothane 1.5% via a Mapleson F system. Oxygen was administered after extubation before transport to the recovery area. All children received oxygen (4 litres/minute) in recovery.

Oxygen saturation was monitored from arrival in the anaesthetic room until the child was ready for collection from recovery, using an Ohmeda Biox 3700 pulse oximeter.

The lowest oxygen saturation recording was noted for each patient and each of the original groups was subdivided into two categories: those whose lowest reading was below 90%, and those above (or equal to) 90%. The opioid groups, B and C, were compared with group A (Chi-squared test).

There is a wide range of regimens for premedication in children that range from nothing at all, to routine opioid and anticholinergic sedation. Correspondence in the anaesthetic literature³⁻⁵ has illustrated the varying opinions on the subject. The degree of sedation in the pre-induction

child is ultimately governed by the preference of the anaesthetist. Other studies⁶⁻⁹ relied on arterial blood samples taken at specific intervals, but this ignores episodic decreases in oxygen saturation, which can only be demonstrated by a continuous technique.

There was no significant difference in the incidence of desaturation between children who had received a non-opioid sedative premedication and those who were unpremedicated or had atropine only.¹⁰ It was thus deemed unnecessary to include a group of unpremedicated children in this study.

The results show that morphine, as a premedicant, causes a significantly increased incidence of low SpO₂ (< 90%) in the recovery phase, with this type of anaesthetic. It is not surprising but it is a factor that needs to be considered when opioids are prescribed as premedication in children.

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Table 1. Lowest recorded oxygen saturation in recovery.

Group	< 90%	> 90%
A	4	26
B	10	20
C	11	19

Does xenon deserve a prominent place in anaesthetic practice?

Dr Boomsma and colleagues conclude that xenon probably deserves a more prominent place in anaesthetic practice (*Anaesthesia* 1990; **45**: 273-8).

However, their study raises a number of comments. The authors conclude that the greater requirement for fentanyl supplementation in the group who received nitrous oxide is indicative of the superior antinociceptive properties of xenon, and dismiss the fact that 1.0 MAC of xenon was compared with 0.7 MAC of nitrous oxide as the sole explanation for this difference. Mindful that the MAC 50 value is not only the concentration at which half the population is asleep, but also the concentration at which half is awake; these young, fit, unpremedicated patients were very lightly anaesthetised indeed, since xenon or nitrous oxide were used as sole anaesthetic agents with relatively modest opioid supplementation. This is perhaps suggested by the increase in blood pressure recorded in both groups, whereas a decrease in blood pressure below

baseline values would more likely be anticipated in young, fit patients more appropriately anaesthetised.

A further consideration neglected in the interpretation of the results is the possibility of the rapid development of tolerance to the antinociceptive properties of nitrous oxide in the group who received this agent.¹

Somewhat more surprising than the differences in the neuroendocrine responses between the two groups is the fact that no patient in either group reported awareness during surgery!

It is perhaps unfortunate that the oft quoted and well remembered MAC values for agents are indeed MAC 50 rather than MAC 95 values. Here then in xenon we have an agent with a MAC 50 value of 71% and where the use of higher concentrations is precluded by the necessity to administer an adequate inspired oxygen concentration; and thus whose use as a sole anaesthetic agent would undoubtedly court the risk of awareness.

Notwithstanding these clinical considerations, widespread clinical use would be untenable even in a semi-closed system at £61 for a one-hour anaesthetic: a figure against which even high flow isoflurane anaesthesia would appear frugal.

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A reply

Thank you for the opportunity to continue the discussion on xenon as an anaesthetic agent and for letting me see the stimulating theoretical comments of Dr K.J. Power.

Whether xenon deserves a more 'prominent' place in anaesthetic practice is not an easy question to answer before this gas is submitted to fair clinical use and judgement of time. Dr Power is concerned about possible awareness during an anaesthetic procedure as we described in our article. Experience shows otherwise and this may also be applied to the reported clinical situation. We agree that the nonsignificant increase of blood pressure may be indicative of 'light anaesthesia', or whatever this term denotes. However, it cannot be disputed that our patients certainly were adequately anaesthetised, to judge from both clinical observation and from the data we reported. One would expect that xenon would need further supplementation for more heroic surgery.

We understand the cardiovascular responses of a patient during a particular anaesthetic, but it is much more diffi-

cult to discuss the anaesthetic state, denoting lack of consciousness, in this context. Firstly, a patient is not asleep during anaesthesia, although Sir Robert Macintosh thought it worthwhile to retain this euphemism for the patients' sake (personal correspondence). Secondly, the MAC estimates of the anaesthetic potency are unreliable in the presence of neuromuscular paralysis. This was the case in our study and is not very different from everyday practice. It may be suggested that, given adequate antinociception, a paralysed patient is less likely to be conscious than when muscle tone is still present. Proprioceptive stimulation of brain is the most reliable sustainer of awakening and wakefulness. That is why gentle shaking has a better chance to awaken someone from deep sleep than a telephone or a knock on the door.

Tolerance to the antinociceptive effects of nitrous oxide may have played a role in our study. However, we were not inclined to make such suggestions because the procedures were of modest duration and such an interpretation of results would have brought forward questions on existence of tolerance to actions of xenon. Such data has not been published, to our knowledge, and any speculation in comparison to the properties of nitrous oxide would be premature, though inviting.

We would like to add that the current cost of xenon need not be a definite obstacle for considering this gas of practical importance to the anaesthetist. Anaesthesia is too inexpensive compared with the extravagant costs of surgical procedures. More importantly, the community of anaesthetists must use and study an agent for some time before final verdicts are credible and xenon should be given the same fair treatment.

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J. RUPREHT

Awareness during Caesarean section

Awareness during general anaesthesia is a constant worry for obstetric anaesthetists and the paper by Bogod *et al.* (*Anaesthesia* 1990; **45**: 279-84) suggests that we are still unable reliably to either detect or prevent it. I believe that anaesthetists have perpetuated an unnecessarily high incidence of this distressing problem by dogmatic adherence to techniques that have little basis in physiological or pharmacokinetic principle.

The routine use of 50% N₂O only in obstetric anaesthesia is quite unnecessary. Pulse oximetry is available to ensure adequate maternal O₂ saturation while allowing the full anaesthetic benefit (and the increased second gas effect) to be obtained from the use of higher concentrations of N₂O. Awareness under anaesthesia is almost unheard of when 66% N₂O is employed; and ventilation with 33% O₂ will prevent hypoxaemia in almost all obstetric patients.

The widespread (and otherwise commendable) practice of protecting the eyes with tape during anaesthesia should be avoided in obstetrics since the pupil reaction to light is usually an excellent guide to the depth of anaesthesia (as described by Guedel many decades ago!). Dare I suggest that this serves as the patient's inbuilt cerebral function monitor and should allow higher initial vapour concentrations and more rational adjustment in the first few minutes of anaesthesia, without any significant risk of uterine relaxation or fetal depression?

Odstock Hospital,
Salisbury SP2 8BJ

D.J. LINTIN

A reply

Thank you for the opportunity to reply to this letter.

The use of 50% oxygen is surely not intended to prevent maternal hypoxia, as suggested by Dr Lintin, but to establish fetal hyperoxia in preparation for the abnormal stresses of delivery by Caesarean section. There is certainly plenty of evidence that babies delivered to mothers whose lungs are ventilated with only 30% oxygen have umbilical vein Po₂ values in the normal (rather than hyperoxic) range, and are disadvantaged on assessment by 1-minute Apgar score.¹⁻³ It would be injudicious to increase the risk to the fetus in this way in order to reduce the likelihood of maternal awareness, especially since raising the inspired concentration of nitrous oxide from 50% to 70% only increases the potency of the anaesthetic mixture by about 0.2 MAC. The same end can be easily achieved without compromising the fetus by Dr Lintin's other suggestion; an initial increase in the inspired concentration of volatile agent. This is surely the better alternative.

The pupillary reflex is one of many clinical signs which can be used as a guide to anaesthetic depth, but which are unfortunately far from infallible. It should be a matter of concern to all anaesthetists, and patients, that a reliable, practical monitor of anaesthetic depth has yet to be discovered.

City Hospital,
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D.G. BOGOD



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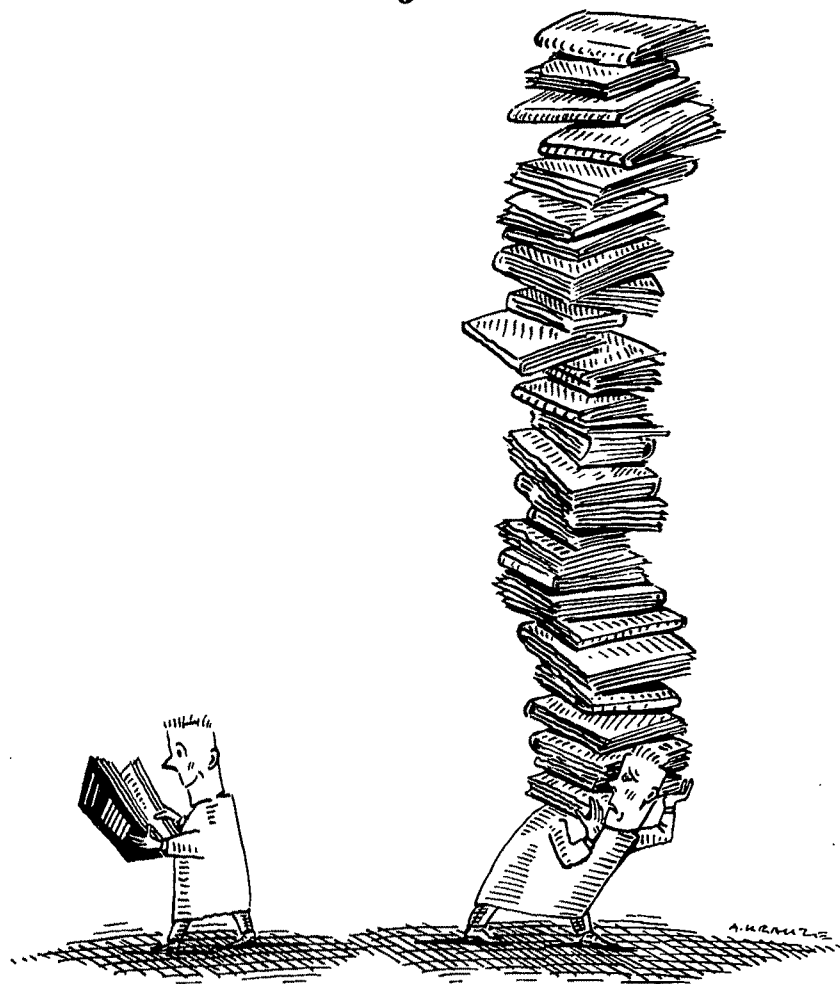
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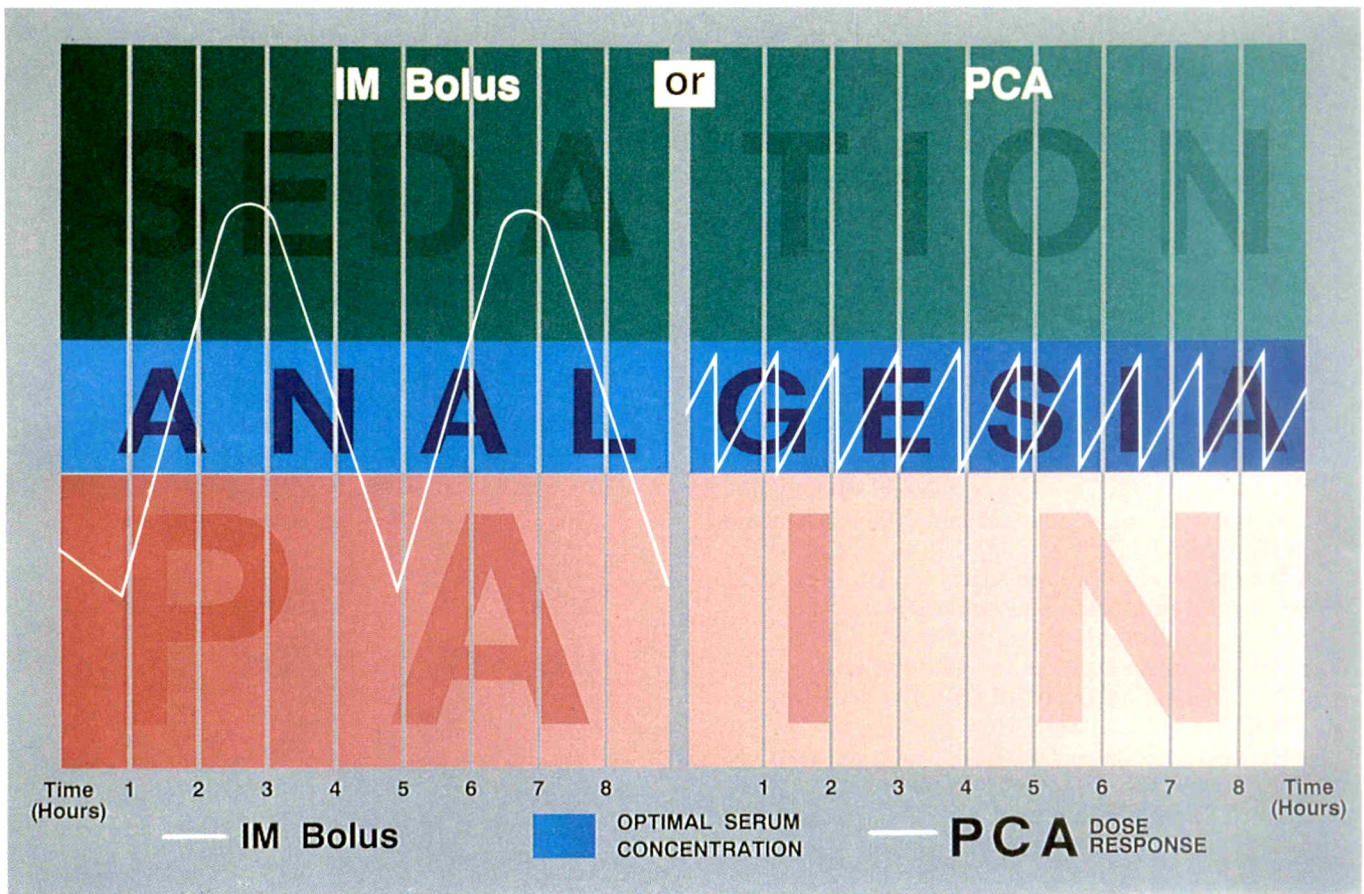
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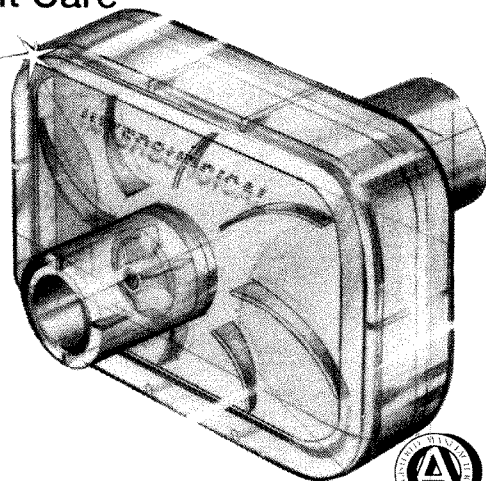
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Crystalloid preloading in spinal anaesthesia

Coe and Revanäs (*Anaesthesia* 1990; **45**: 241-3) reported a study of preloading with Ringer's acetate solution for spinal anaesthesia in the elderly. A total of 60 patients were studied. The incidence of significant hypotension (a decrease of 25% or more in systolic blood pressure) was found to be 25% in a group who had received no preloading, 24% in a group who had received 8 ml/kg and 32% in a group who had received 16 ml/kg. It was concluded that 'crystalloid preloading, with volumes in common use, is not effective in the reduction of the incidence of hypotension after spinal anaesthesia'.

This is possible, but the small number of patients studied prevents such a conclusion being drawn. There was a 7% difference in relative risk of hypotension between the unpreloaded patients and the group who had received the larger preload volume. However, the numbers studied are so small that the 95% confidence interval for this difference extends from -21% to +35% (using the equation given by Colton).¹

Sackett *et al.*² published tables that allow the adequacy of the sample size in this sort of study to be assessed. It appears from these tables, that Coe and Revanäs can be confident (at the 0.05 level) that, if crystalloid preloading actually halved the relative risk of hypotension, they would have detected a significant difference between their groups. Lesser effects attributable to preloading might well have gone undetected by their small study.

If crystalloid preloading does, in fact, reduce the relative risk of significant hypotension by a quarter or a third (without increasing morbidity), it is a valuable component of spinal anaesthesia. Coe and Revanäs are unable to exclude such an effect, and therefore should not, at present, persuade us to alter our clinical practice. Their conclusions are couched in suitably guarded terms, but perhaps some quantification of the relevance of their results (such as presented here) should have been included in their paper.

Ninewells Hospital,
Dundee DD1 9SY

G.L. HUTCHISON

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We were interested to read Drs Coe and Revanäs paper on the management of spinal anaesthesia in the elderly (*Anaesthesia*, 1990; **45**: 241-3) and agree with their conclusion that crystalloid preloading does not reduce the incidence of concomitant hypotension. We consider, however, that the methods of reducing its occurrence below 30% are already well-known, and we are therefore surprised that it was necessary to subject so many elderly patients to the risks of hypotension and possible harm.

The blocks for lower abdominal and limb surgery were excessively high and this was why they were associated with a 25-30% decrease in blood pressure. No patient who had a block to T₈ or below experienced this decrease in blood

pressure. We wish to comment on the methods of this study.

The factors that determine the height of a subarachnoid block are the baricity of the solution, site of injection, patient posture, volume and rate of injection, and age.¹

Isobaric bupivacaine produces a wide range of unpredictable spinal blocks and it is not marketed for intrathecal use.² Hyperbaric bupivacaine produces a narrower, and more reliable, range of blocks above or below the umbilicus.³ It is known that injection at the L₂₋₃, rather than the L₃₋₄ interspace, is associated with an average level of anaesthesia four segments higher and a greater incidence of arterial hypotension.²

The authors do not record the posture of their patients. Greene states that if the cardiac output remains constant, the mean arterial blood pressure decreases 12-15% in normovolaemic patients because of the decrease in total peripheral resistance and that further decreases are caused by inadequate venous return.⁴ If the hypotension reported in the study reflected inadequate venous return it could have been corrected by early use of leg elevation or the head-down position, without changing the height of the blocks performed with an isobaric solution.⁵ Small volumes of solution injected slowly produce the most predictable blocks.⁶ The study employed relatively large volumes.

Increasing age of the patient is associated with a higher level, a faster onset and a more profound block than in younger subjects.⁷ It is often suggested that patients over 75 years differ in many ways, including their autonomic function, from younger groups.⁸ This study did not elucidate this since the groups contained both men and women aged 60 to 90 years and did not differentiate between the merely old, and the elderly.

Walsgrave Hospital,
Walsgrave,
Coventry CV2 2DX

R.J. ELTON
R.S.C. HOWELL

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A reply

Thank you for the opportunity to reply to Dr Hutchison's letter: I am grateful to him for his statistical calculations but must disagree with his conclusions. He states that a halving of the relative risk of hypotension would be required to show a significant difference between the treatment groups. We found minimal differences between the preload groups with respect to the incidence of hypotension. We also examined the level of block at which hypotension first occurred, and the proportion of patients at each block level that became hypotensive in the three patient groups. There was no hint that preloading was effective. It is most unlikely that any major benefit of preloading went unnoticed.

The reason for the wide 95% confidence intervals for the hypotension incidences was that the risk of hypotension in this study was relatively small (of the order of 30%). A prophylactic measure given for a complication suffered by only a minority of those who receive it must be highly effective to be considered useful. This is especially so if side effects can be expected from it¹ and if the complication is easily treated. We conclude that the study suggests that routine crystalloid preloading before spinal anaesthesia in the elderly is not a useful procedure.

It is also a pleasure to note that Drs Elton and Howell agree with our conclusions. There remains disagreement about the value of clear fluid preloading as Dr Hutchison's letter and inspection of current textbooks shows.²⁻⁵ The treatment threshold was set deliberately low in this study and the patients were continuously supervised by an anaesthetist with effective treatment available. Serious hypotension was not allowed to develop. The risk to which these patients were exposed was no different to routine cases on

the same lists. It was thus neither excessive nor unnecessary. Drs Elton and Howell suggest that our block levels were too high. Our target level was T₇ in 20% of cases, to cover cord traction during hernia repair. Other similar work shows a far greater percentage of high thoracic blocks.⁶

The authors' observations on the factors which affect block height are interesting. Our intention was not to produce low or predictable blocks but to assess the effects of preloading, so the comments are not relevant. All patients were placed supine after lumbar puncture.

Walton Hospital,
Liverpool L9 1AE

A.J. COE

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Awareness during anaesthesia

The Editorial by Professor Aitkenhead, (*Anaesthesia* 1990; **45**: 351-2) will be of great assistance to anaesthetists who are confronted by the problems of awareness under anaesthesia. Speakers at two seminars at 9 Bedford Square mentioned the need to improve the care of patients who experience awareness, and the need to support the anaesthetist concerned, through an experience which is very traumatic. Seventy anaesthetists have attended these sem-

inars, so we should be able to ensure that 'a consultant with a special interest in awareness' would be available to advise anaesthetists who have to deal with cases of awareness.

The writer is prepared to help such an anaesthetist should any reader experience difficulty.

Lewisham Hospital,
London SE13 6LH

J.M. CUNDY

Monitoring during sedation

The use of regional anaesthesia has now become an accepted method of practice for surgery of the pelvis, lower abdomen and lower extremities and it is, in fact, the anaesthesia of choice in many cases. Most anaesthesiologists consider it to be good practice to add some degree of intravenous sedation by use of fentanyl, diazepam, midazolam, thiopentone or droperidol in subanaesthetic doses.

This induces a sleeplike state with no verbalisation and improves patient comfort, but exposes the patient to various risks including ventilatory depression, loss of airway reflexes and haemodynamic instability.¹ These may all lead to hypoxia, hypercarbia and eventual cardiac arrest if left untreated.² The usual monitoring techniques used in this type of anaesthesia include an E.C.G., blood pressure measurement, precordial stethoscope and pulse oximetry.³

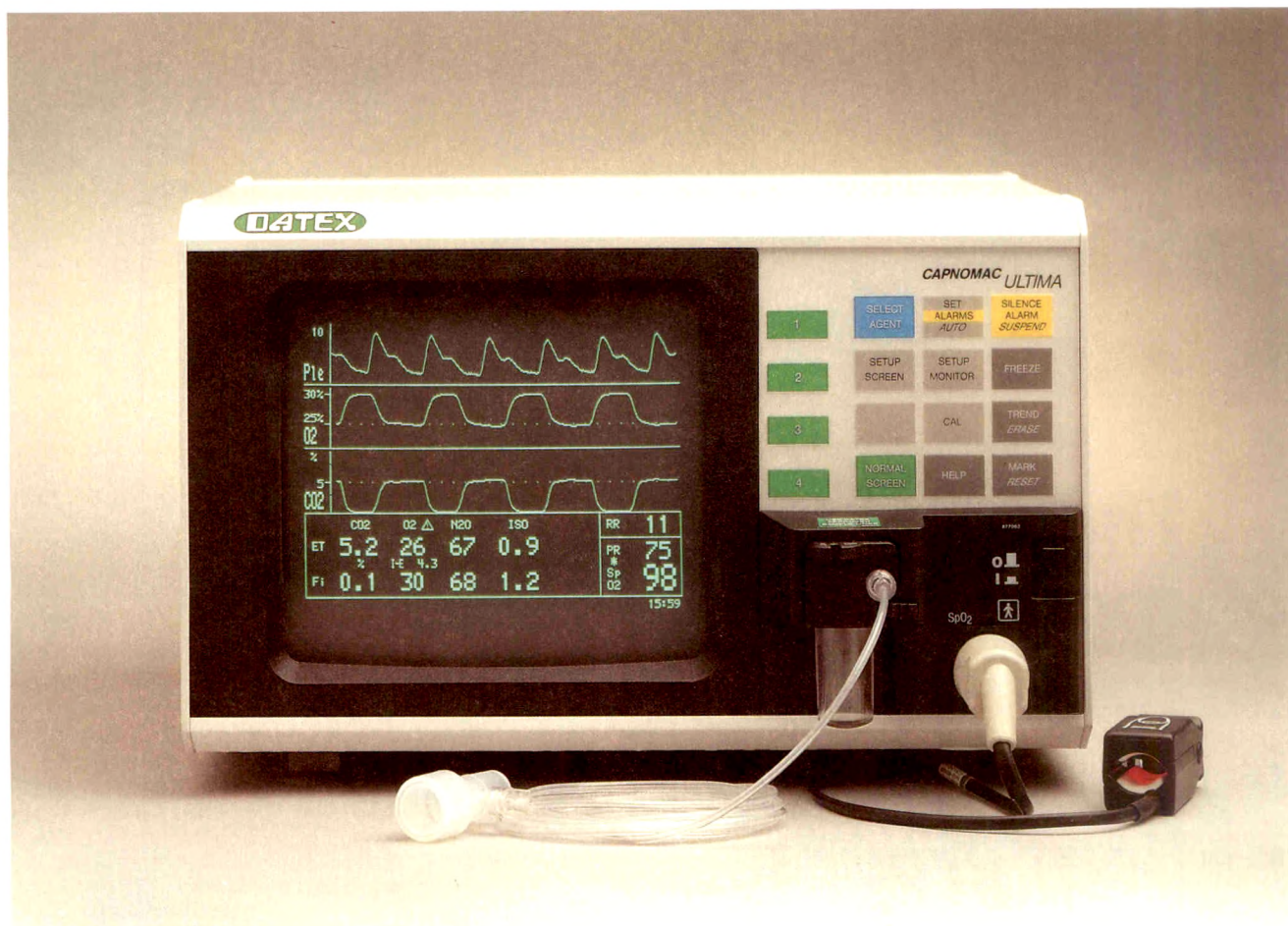
All this information is important, but may not indicate immediately the onset of ventilatory depression in the sedated patient. The first signs of ventilatory depression are reduction in both rate and volume of ventilation, but in

apnoea, a period of at least 2 minutes is required before there are significant changes in oxygen saturation, displayed by oximetry.

This latent period may be critical, particularly in a patient who is already an increased risk or in a compromised category. Assessment of ventilation in a spontaneously breathing, asleep, patient is performed normally either by visual means, i.e. watching the patient's chest movement, or by use of a precordial stethoscope. We have devised a method of monitoring by use of capnography with a Datex Cardiocap Monitor, which we consider to be both more accurate and sensitive than those previously used.

Immediately after inception of the regional block, we supplement the patient's inspired O₂ by means of an open mask (Laerdal Pocket Mask) Fio₂ 0.4, 2-4 litres/minute (Fig. 1). The capnograph gas sampling tube is corrected to the mask outlet (Fig. 2), which enables us to record on the Cardiocap both the number of breaths per minute numeri-

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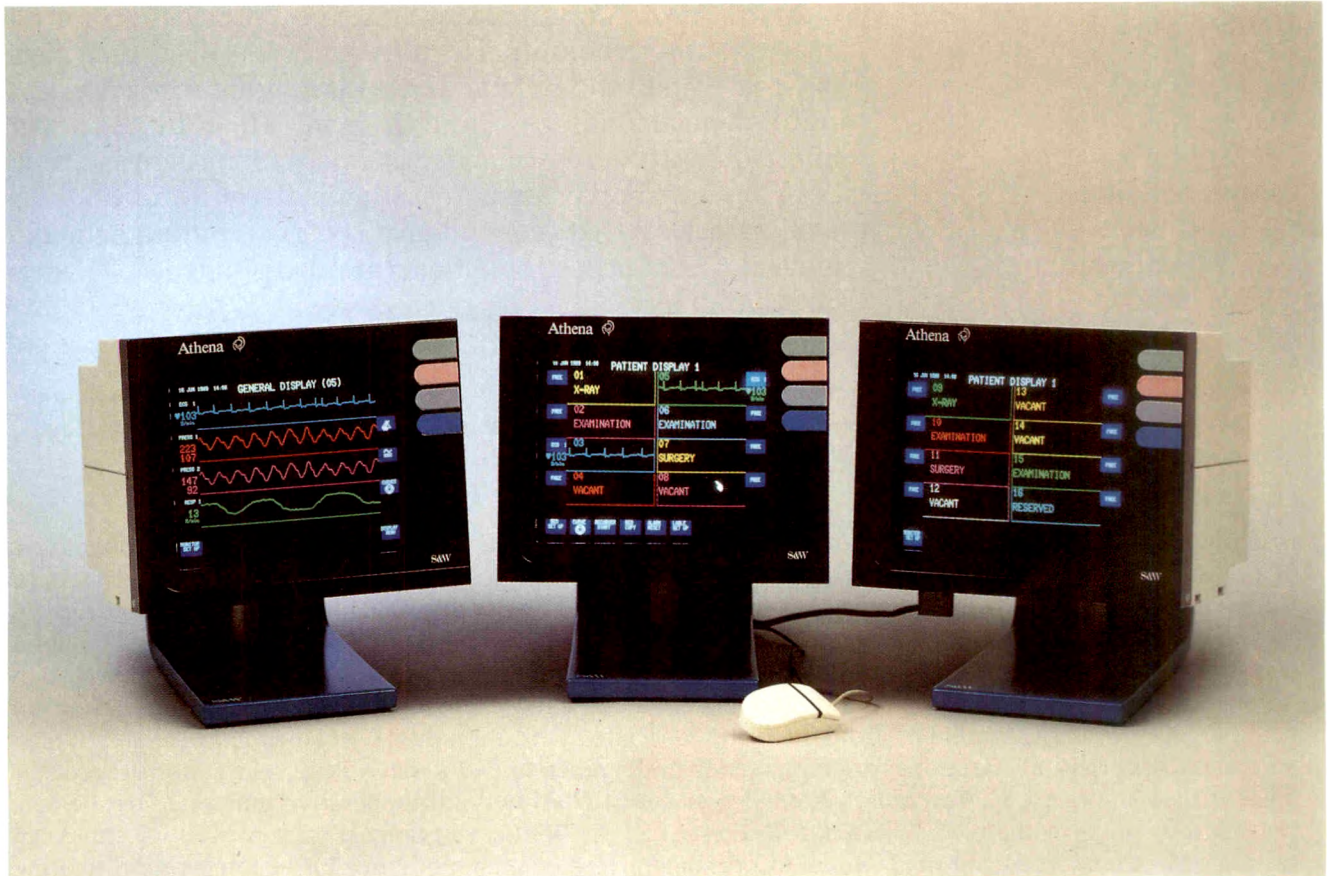
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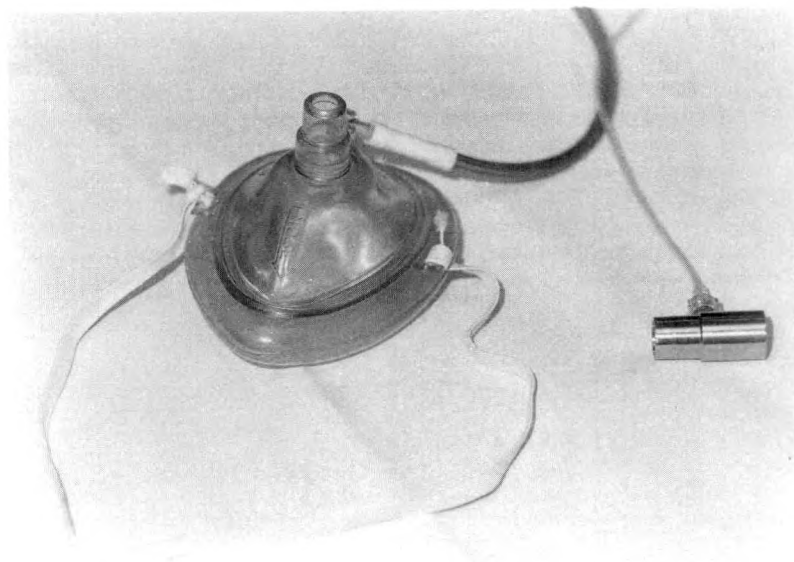


Fig. 1. Laerdal open mask with supplementary oxygen supply and capnograph sampling tube.



Fig. 2. Laerdal open mask attached to a patient.

cally and to record the CO₂ waveform graphically. Any changes in the *quality* of ventilation will be detected immediately on a breath-to-breath basis.⁴

The use of capnography in a spontaneously breathing patient is a more sensitive method of detection of early changes. Other variables are still within normal limits. We have used this technique for approximately one year (400 cases) and are able to recommend its effectiveness.

Nahariya Government
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Management of supraventricular tachycardia in septic patients

Supraventricular tachycardia (SVT) may complicate the management of patients in septic shock. Many factors contribute to the evolution of SVT in septicemia including fever, circulating endotoxin and peripheral vasodilatation. A standard treatment in uncomplicated SVT is verapamil.

Its actions on the conducting system are beneficial, but myocardial depression and vasodilatation may result in severe hypotension.

We evaluated 19 postoperative patients with septicemia and vasodilatation. Each patient developed SVT after sur-

gery for cancer and showed signs and symptoms of septicaemia including fever (38.4–39.0°C), leucocytosis tachycardia and hypotension (mean arterial pressure 20–30 mmHg less than before operation). When SVT started the blood pressure declined an additional 10–40 mmHg. The first group of nine patients were treated with verapamil alone. The second group consisted of 10 patients who were treated with phenylephrine and verapamil.

Group 1 patients were given verapamil in increments of 2.5 mg every 5 minutes to a total dose of 10–15 mg. Verapamil precipitated an immediate decrease in blood pressure in all nine patients. Four patients converted to a sinus rhythm, three required cardioversion and digoxin, and two suffered cardiac arrest immediately after the verapamil-induced hypotension.

The second group of patients received phenylephrine and verapamil. Five patients responded to phenylephrine 100–200 µg and verapamil 5 mg while the other four patients required phenylephrine 300–500 µg and verapamil 10 mg for conversion to a sinus rhythm.

The tachycardia in septic patients may be in response to vasodilatation, increased catecholamines, fever or endotoxins. Verapamil slows atrioventricular conduction, depresses the myocardium and causes peripheral vasodilatation. The effect on the conducting system is useful in the termination of SVT, but the myocardial depression and vasodilatation are undesirable side effects in hypotensive patients. Drugs which cause peripheral vasoconstriction, such as phenylephrine, are sometimes effective in slowing the heart reflexly and in conversion of SVT to a sinus rhythm. This study demonstrates that phenylephrine prevents hypotension and potentiates the therapeutic effect of verapamil in SVT.

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Trauma to epidural veins: the role of posture

The sequelae of damage to the epidural venous plexus during the insertion of a lumbar epidural catheter can, at best, be an inconvenience, and require reinsertion at a different site, or at worst a major catastrophe with the formation of a spinal haematoma. Commonsense and a knowledge of anatomy leads one to conclude that this valveless plexus becomes engorged when a patient moves from the lateral to the sitting position. One would further assume that this would make damage to these vessels during the insertion of an epidural catheter more likely. A simple two-part study was conceived to test this hypothesis.

Firstly, the records of 1060 obstetric epidurals inserted by six anaesthetist colleagues were studied. Three of these anaesthetists inserted all of their epidurals with the patient in the sitting position, and the other three with the patient in the lateral position. The incidence of epidural vein trauma (defined as either blood freely aspirated through Tuohy needle or free back flow along the catheter) was 6.8% in the sitting group (573 patients) and 5.3% in the lateral group (487 patients). This difference was not significant.

Secondly, I studied my own clinical practice. Two hundred and nineteen consecutive obstetric epidurals were inserted with the patient in the sitting position and the next 86 with the patient in the lateral position. The incidence of epidural vein trauma (as defined earlier) of the first 20 insertions in each patient position were compared with the subsequent insertions in that position and secondly, the overall incidence in the sitting versus lateral positions. The results are given in Table 1.

The incidence of vein trauma was significantly higher during the first 20 insertions in both positions when com-

Table 1. Incidence of bleeding in relation to position of the patient.

	Number bleeding	%	Overall %
First 20 sitting	5	25**	6.8
Next 199 sitting	10	5**	
First 20 lateral	5	25*	10.5
Next 66 lateral	4	6*	

**p < 0.01; *p < 0.05.

pared with the subsequent insertions. However, there was no significant difference in the overall incidence between sitting and lateral positions.

There appears to be no difference in the incidence of trauma to the epidural vein with the patient in the sitting or in the lateral position. However, a change in operator technique from one position to the other does appear to increase the incidence. This is presumably because a temporary difficulty in locating familiar landmarks led to the needle straying towards the epidural veins in the lateral parts of the space. It seems prudent advice therefore, to recommend the establishment of one's own technique and, where possible, to stick to it.

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Convulsions after cocaine and propofol

Most of the reported incidents of opisthotonus and grand mal convulsions associated with propofol have involved either a history of investigation for epilepsy, a family history of epilepsy or diagnosed epilepsy.^{1–4} All of the reactions in patients without any epileptic history included the use of intravenous alfentanil,^{5–7} with the exception of one patient.⁸ The reactions seen in the first group begin with an opisthotonic posture and lead to repeated grand

mal convulsions. The second group exhibit an opisthotonic posture that may proceed to one episode of convulsions.

This report is about a case of convulsions after the use of propofol, which was neither associated with the use of alfentanil nor a history of investigation for epilepsy nor a familial tendency towards epilepsy. A fit 23-year-old male presented for a septorhinoplasty for cosmetic reasons. There was no history of head injury. He had no significant

personal or family history and had never received medication. He had one previous general anaesthetic, which was uneventful, for an appendicectomy 10 years before. Premedication was with papaveretum 15 mg and hyoscine 0.3 mg. His trachea was intubated with a 9-mm RAE tube, after propofol 200 mg and suxamethonium 75 mg. Ten percent cocaine paste was applied to the nasal mucosa. He was allowed to breathe spontaneously a nitrous oxide, oxygen mixture with 2% isoflurane. The procedure lasted 1 hour and 35 minutes, and blood pressure, pulse, ECG and oxygen saturation were monitored during the operation. He woke up after the procedure on the way to recovery and 10 minutes after arrival developed a dystonic reaction which proceeded to a generalised convulsion. He did not need reintubation and the convulsions did not recur.

He was drowsy afterwards and had bilateral upgoing plantar responses together with ataxic nystagmus. He made a good recovery and was discharged home without sequelae.

Cocaine is absorbed from the nasal mucosa and reaches peak blood levels between 15 and 60 minutes after administration; it persists in the plasma for 4 to 6 hours.⁹ His pulse rate, 70 beats per minute, and systolic blood pressure, 130 mmHg, had remained steady.

There was no cardiac arrhythmia, postoperative nausea or vomiting suggestive of cocaine toxicity.⁹ Cocaine is

known to interact with antihypertensive drugs by adrenergic neuron mechanisms. It is still not clear whether this convulsion was from propofol alone or the result of interaction with cocaine.

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Electro-acupuncture and postoperative emesis

It is gratifying to learn that, in well designed studies, Dr Ho and colleagues (*Anaesthesia* 1989; **45**: 327-9) have confirmed our published findings¹⁻⁴ that, under appropriate circumstances stimulation of the P6 (Neiguan) acupuncture (ACP) point can have a positive antiemetic action. However, there are certain points of difference from our results which are worthy of note.

Nausea is indeed a subjective sensation, but it is easy to elicit. Had it been included in Dr Ho's study, then the efficacy of ACP might have been more marked and perhaps the benefit from transcutaneous electrical stimulation (TCES) might have been significant. We found that reduction in nausea by ACP was greater than that of vomiting.¹ The beneficial effect of phenothiazine, such as prochlorperazine is, in contrast, mostly in reduction of vomiting. We notice this particularly after chemotherapy, where a phenothiazine, while minimising emesis often leaves patients with a distressing feeling of nausea.

The same apparatus (TENS) can be used for stimulating both the ACP needle and the surface electrode. In a 'frequency finding' study² we used 10, 20, 50 and 100 Hz and concluded that 10-20 Hz produced maximum benefit, while 100 Hz was mostly ineffective. Likewise, there was no benefit from increasing the duration of stimulation from 5 to 15 minutes.

There is a slight advantage in using the dominant arm,⁵ but this is not important since we found only 75 out of 1000 patients questioned claimed to be left handed. In our current studies on 'self administration' of TCES with cancer chemotherapy, many right-handed patients could not use the TENS machine with their left hand, but this has not distracted from the benefit.

It is noted correctly that one investigation⁶ showed no antiemetic effect when ACP was administered during anaesthesia. We have reported a similar finding.⁷ This cannot be a psychological effect since the stimulation of a nonacupuncture point (dummy) produced no benefit,^{1,3} in patients who were given the same explanation of the study as those having P6 stimulation. We believe that to be

effective, ACP should be administered before the emetic stimulus. (Others have shown that this applies to conventional antiemetics in chemotherapy).⁸ When administered before the emetic stimulus (nalbuphine premedication) only 22% patients had either nausea or vomiting, compared with 60% when given immediately before or after the induction of anaesthesia.⁹ the control (no treatment) incidence was 68%. These findings can be reconciled with those of Ho and colleagues, even though they performed ACP after administration of the fentanyl, which is a potent emetic stimulus. The emetic effect of fentanyl at the time of the ACP would be counteracted by the powerful antiemetic effect of halothane.¹⁰⁻¹² ACP would be inhibiting its antiemetic action by the time the halothane wears off and fentanyl could exert its emetic action.

Could the failure to demonstrate a beneficial effect from transcutaneous electrical stimulation be because of the brevity of antiemetic action exhibited by all noninvasive methods?^{2,13,14} The antiemetic action of either acupressure or TCES cannot be depended on for more than 2 hours: if the stimulation had been repeated at this time the results might have been better. We have found in cancer chemotherapy that pressure on the stud of the commercially available 'Sea Bands' every 2 hours will prolong the beneficial effects of ACP for up to 24 hours.¹⁵ Wearing the band alone (as recommended by the manufacturer's for travel sickness) is not adequate.

The theory discussed by the Taiwan workers is interesting, but probably only partially correct. The release of endogenous endorphins is only reported with high frequency ACP, as used for pain relief,^{16,17} but this effect can be antagonised by naloxone,¹⁸⁻²⁰ as can morphine analgesia. However, the emetic action of morphine can also be abolished by naloxone. The antiemetic action of low frequency ACP (30 Hz by Ho *et al.* 10-20 Hz by ourselves) may involve another, as yet unidentified endorphin-like compound.

Finally, while agreeing that the application of acupuncture can be cumbersome and time consuming, one must not

dismiss it as without application in anaesthesia. Manual rotation of the needle, which is as effective as electro-acupuncture^{2,3} requires no apparatus other than a needle. It is completely nontoxic and in over 2000 applications we have only once struck the median nerve. We are trying to perfect a portable apparatus for transcutaneous stimulation which can be attached to the forearm in a manner similar to Sea Bands.

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Problems associated with the introduction of different equipment

Recently, our department introduced a new epidural pack as part of an effort to reduce costs; previously, a variety of equipment from different manufacturers was used. The changeover to a complete pack from one manufacturer (Everett) has been associated with a number of problems.

An incident occurred in which the epidural needle sheared off at the joint between the needle shaft and the hub, whilst the needle was being inserted into a patient's back; this required the removal of the needle shaft with the aid of a pair of forceps, but no injury to the patient resulted from this incident.

Two incidents have occurred in which the 'needle'/Luer lock device used to connect the catheter to the filter has broken at the junction between the 'needle' and its hub. These have exposed the patients to the possible risk of puncture wounds from the 'needle' as well as the potential infection risks that may result from exposure of the catheter to air.

Several of our staff have noticed that the epidural needle point is somewhat sharper than that of our previous choice (Portex) and that the dural tap rate rose by several percent in the initial period after introduction of this set.

It is possible that there is no association between these observations, but we consider that the needle point is a potential source of inadvertent dural taps and that there is almost certainly going to be an increase in the dural tap rate when an established set of equipment is replaced by new equipment.

It must be said that no injury to any patient occurred as a result of these incidents, although the potential for morbidity was present.

These problems will be reported to both the manufacturer and the relevant section of the Department of Health.

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The use of transtracheal cannulation after difficult intubation

A 63-year-old man presented with a frontal lesion and required a craniotomy. He was mildly hypertensive and somewhat obese (110 kg) but was otherwise well. A general anaesthetic 20 years ago was associated with some difficulty in tracheal intubation, although a precise history was not obtainable. Inspection of the fauces in the manner

described by Mallampati^{1,2} was unremarkable. The patient was premedicated with atropine 0.6 mg. Venous access was secured and the patient was pre-oxygenated. Anaesthesia was induced slowly with thiopentone 300 mg, the lungs were found to be easily ventilated via a facemask, and suxamethonium 100 mg was administered. The tip of the

epiglottis only was visible at laryngoscopy, and atracurium 50 mg was given to facilitate this. A 9.0-mm flexometallic (Portex) tracheal tube was passed after some difficulty into the trachea over a long bougie. The patient underwent uneventful craniotomy which lasted 2 hours. Neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.2 mg. Spontaneous ventilation was established and the patient opened his eyes to command. Extubation was deemed desirable to facilitate neurological observations. We considered that the airway should be secured prophylactically in view of the difficulties encountered during intubation because of the potential need to acquire control over the airway in the postoperative period. A 13 G VBM cricothyroid cannula (VBM Medizintechnik, West Germany) was inserted into the trachea via the cricothyroid membrane. The tracheal tube was withdrawn and the patient transferred to the recovery area and thence to the neurosurgical intensive care unit for overnight observations. A Sander's injector was placed by the patient's bedside and the on-call staff instructed in its use. The necessity for ensuring an unobstructed path for expiration was emphasised.

The use of transtracheal cannulation inserted under local anaesthesia in cases of anticipated difficult intubation was

described³ as has its use after operation when access to the airway was rendered difficult by jaw wiring.⁴ We conclude that a further indication for transtracheal cannulation is in cases of unexpected difficult (but successful) intubation when access to the airway may be required.

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Laryngeal mask and anaesthetic waste gas concentrations

The incidence and magnitude of waste anaesthetic gas leakage during laryngeal mask (LM) anaesthesia is reported. We measured nitrous oxide levels continuously from the breathing zone of the anaesthetist during LM mask anaesthesia in seven healthy, adult patients with a Miran 1A general purpose infrared gas analyser, (wavelength was 4.5 μ m, pathlength 14.25 m and response time 1 second). The sampling hose was attached 0.3 m behind and

0.3 m above the patient's head, a point considered to be within the anaesthetist's breathing zone. All patients received a standard premedication and induction with thiopentone and breathed spontaneously during the anaesthetic. The fresh gas flows were 5 litres/minute 70% nitrous oxide in oxygen through a circle system with soda lime absorber. Halothane was used in all the cases.

The anaesthetic room had a volume of 35 cu m and the

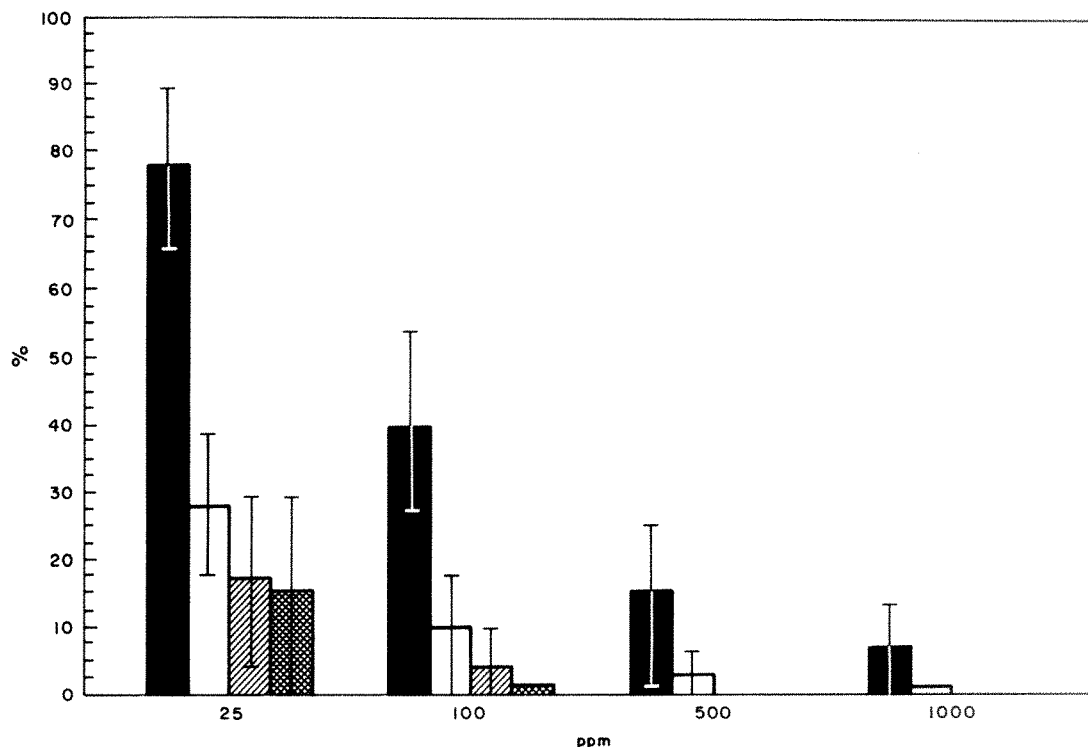


Fig. 1. Fraction of total anaesthetic time, (mean, SD) during which period the nitrous oxide concentrations were above the limits mentioned. ■, mask anaesthesia, $n = 5$, total anaesthetic time (TAT) 386 minutes; □, mask anaesthesia with close scavenging (CS), $n = 6$, TAT = 313 minutes; ▨, intubation anaesthesia with CS, $n = 6$, TAT = 407 minutes; ▩, LM anaesthesia with CS $n = 7$, TAT = 334 minutes.

Table 1. Nitrous oxide levels in the anaesthetist's breathing zone during seven anaesthetics. (Close scavenging in use when LM is not in use.)

Patient's details		Measurement	Peak	Mean
Age	Sex	time (minutes)	level ppm	level ppm
24	M	52	25	3
59	M	51	27	4
46	F	55	37	4
50	F	61	230*	6
34	M	33	15	4
39	M	47	32	6
40	F	35	29	5

*Manual ventilation was performed for a short time to confirm the position of the LM. The value shows evidence of leakage of anaesthetic gases during positive pressure ventilation.

room ventilation was 560 cu m/hour, approximately 16 air changes/hour. The active scavenging system at the waste gas outlet of the anaesthetic machine had a capacity of 25 litres/minute. A close scavenging device, designed to evacuate at a rate of 27 cu m/hour was placed within 20 cm from the patient's mouth for the duration the LM mask was not in position. The patient details and the mean and peak concentrations of nitrous oxide are given in Table 1.

Laryngeal mask airway for inadequate reversal

Another use of the laryngeal mask airway (LMA) is described. A 27-year-old female previously healthy who weighed 80 kg was scheduled for laser laparoscopy. She was premedicated with temazepam 30 mg orally 2 hours before operation. Anaesthesia was induced with alfentanil 500 µg, vecuronium 8 mg, and propofol 150 mg. The trachea was intubated easily and her lungs ventilated to normocapnia with 33% oxygen, 66% nitrous oxide and 1% enflurane. Blood pressure, electrocardiograph, oxygen saturation and end-tidal carbon dioxide were monitored. Laser treatment was started after laparoscopy. Five minutes later the laser machine developed some trouble and, since there was no prospect of immediate repair, the surgical procedure was abandoned. Muscle paralysis, 30 minutes after vecuronium was reversed with glycopyrronium 0.5 mg and neostigmine 2.5 mg. She appeared to be awake and breathing adequately; oxygen saturation was 96% and she coughed on the tracheal tube, so she was extubated. A few minutes afterwards her oxygen saturation decreased to 80% and peripheral cyanosis developed even with 100% inspired oxygen. She had a tracheal tug and jerky movements which suggested incomplete reversal. A nerve stimulator was attached and T4:T1 was found to be approximately 50%. Other causes of inadequate ventilation were excluded. It was decided to reintubate the trachea but this proved difficult because the cords were moving and she was restless. A size 3 LMA was introduced without diffi-

Figure 1 shows the nitrous oxide levels obtained under similar circumstances using tracheal intubation, ordinary mask, and with and without close scavenging systems. These measurements clearly show that LM mask results in minimal theatre pollution during spontaneous ventilation and is comparable to values obtained after tracheal intubation. Higher levels of nitrous oxide were obtained during periods of positive pressure ventilation, as indicated by the peak level in case 4; levels were transiently higher before the introduction and after removal of the LM mask. We recommend the use of a close scavenging system during these periods to reduce the levels to within the 25 ppm limit suggested by NIOSH.¹

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culty and ventilation was assisted with 33% oxygen and 67% nitrous oxide. The oxygen saturation improved within minutes and she started to breathe adequately after 5 minutes. The laryngeal mask was subsequently removed without any further airway problem.

Extubation after reversal of neuromuscular blockade is usually straightforward but occasionally a dilemma can arise. Incomplete reversal is usually managed by following 'The incomplete reversal drill'.¹ The drill involves improving oxygenation by IPPV with a mask and oxygen, by assessing the degree of incomplete reversal, by supplementary doses of reversal agents (if T4/T1 ratio < 50%), reintubation and ventilation with or without relaxant or sedative and by excluding overdose of inhalational agents, opioid, interactions of drugs with relaxants, myasthenic state etc. This drill was followed in the above case except that the LMA was used in place of the tracheal tube without the need for relaxant or sedative, and thus prolonged ventilation was avoided.

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Supplementary oxygen and the laryngeal mask airway

We have been using the Brain laryngeal mask airway in our hospital for several months. We leave the airway in place in the recovery ward until the patient has protective reflexes, but have not found a completely satisfactory way to give additional oxygen therapy to patients with laryngeal masks in place in our recovery ward. Ordinary disposable oxygen

masks are difficult to position over the connector; anaesthetic systems require an anaesthetic machine that may not be available to every patient on the recovery ward and usually can only give 100% oxygen. We have constructed a venturi T-piece system out of readily available items of disposable equipment. The venturi from a Ventikask (Air-

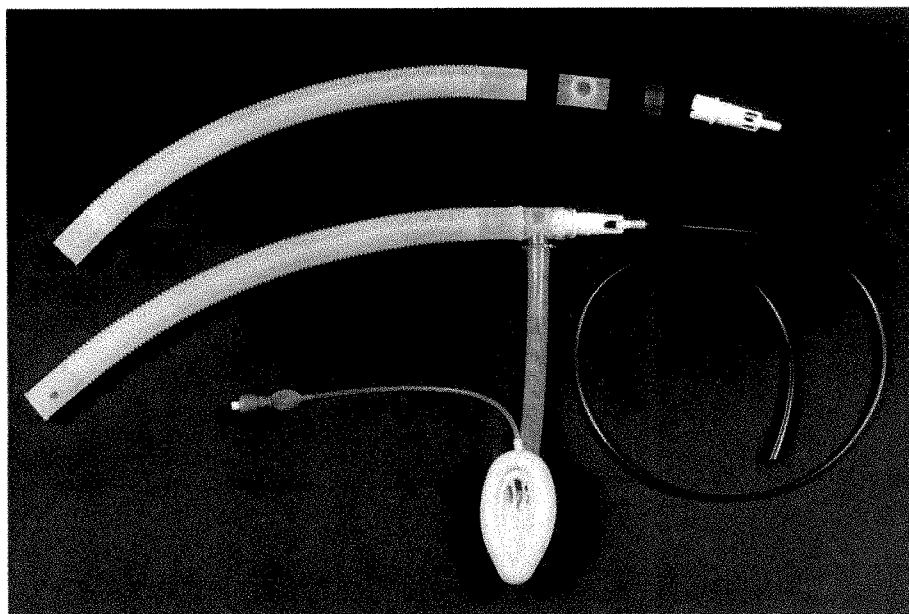


Figure 1. Assembly of components for supplementary oxygen to laryngeal mask airway.

Table 1.

Input O ₂ flow litres/minute	28% venturi measured oxygen %	Total flow (calculated) litres/minute	35% venturi measured oxygen %	Total flow (calculated) litres/minute
2	34	10.8 (12.15)	50	** (5.44)
4	34	23.2 (24.3)	44	9.4 (13.7)
6	34	36.2 (36.4)	44	17.0 (20.6)
8	34	47.0 (48.6)	44	26.0 (27.4)

** Flow too small to measure. Total flow = $\frac{0.79 \text{ O}_2 \text{ flow}}{\text{Fractional oxygen concentration} - 0.21}$

shields Vickers) including the plastic collar cut from the mask is inserted into the side of a Thermal Humidifying Filter for tracheostomies (Portex) from which the paper has been removed. Forty centimetres of disposable anaesthetic tubing is attached to the other side with holes punched at the exit to prevent obstruction (Fig. 1).

We have investigated the performance of this device at various gas flows and found that all the flow meters we connected into this system caused back pressure on the venturi and changed the entrainment ratio. We have, therefore, calculated the flow from the measured oxygen concentration for a 28 and 35% venturi as well as directly measuring the flow with a Wright Respirometer (Table 1).

Theoretically the fresh gas flow to a T-piece should be two to three times the patient's minute volume to prevent rebreathing, so a total flow of 10 to 15 litres/minute should be adequate for the average patient.

We then investigated the minimum oxygen flows required to prevent rebreathing in five patients who were

recovering from anaesthesia by CO₂ analysis, sampled from between the airway and the T-piece, with a 28% venturi, and found that at oxygen flows greater than 2 litres/minute inspiratory carbon dioxide was zero in all patients. Thus for the average patient we use oxygen flows of 4 litres/minute with the 28% venturi and 6 litres/minute with the 35% venturi.

This device is easy to use, has a very low resistance to respiration, gives a fixed inspiratory oxygen percentage at an economical oxygen flow, is light-weight so does not drag on the airway and is safe even if there is a fresh gas supply failure as the patient can breath air via the holes in the venturi.

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The use of the laryngeal mask airway in primary anaesthesia

We were anaesthetists to a field surgical team in Belize recently and have some experience with the laryngeal mask airway (LMA). We evaluated the use of the LMA in field conditions by nonanaesthetists who were given a single

demonstration. The results of successful insertions of the LMA per attempts were: Dental officer 12/12; Dental officer 6/6; ODA 2/2; ODA 2/2, and Certified Registered Nurse Anaesthetist 3/3.

The first dental officer had recently undergone the Anaesthetic Support Resuscitation Officer (ASRO) Course. This is designed to train dental officers for their war role as anaesthetic assistants. He attempted 20 tracheal intubations during that course: he passed six tubes easily, four with difficulty and failed in the remaining 10.

We conclude that the LMA is an easier device to use and its role in primary anaesthesia¹ must be considered especially for the appropriate elective procedures or in failed intubation.²

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The oesophageal detector device and the laryngeal mask

We have applied the oesophageal detector device (ODD)¹ to 100 consecutive placements of the laryngeal mask airway (LMA) in patients undergoing elective urological and breast surgery. There were 72 female and 28 male patients; ages varied from 17 to 82 years. Anaesthesia was induced with thiopentone or propofol and deepened with a volatile agent. An LMA was placed in the recommended manner, the cuff inflated and a standard ODD test applied.² Muscle relaxants were not used. The breathing system was connected and the patient observed for evidence of airway obstruction (stridor, tracheal tug, paradoxical breathing).

The ODD in four patients was positive (bulb did not refill) and the airway proved to be completely obstructed (no stridor, pronounced tracheal tug and paradox); in 17 patients the ODD was equivocal (the bulb refilled slowly). The airway was clinically partially obstructed in these patients, but improved as they became more deeply anaesthetised. A negative ODD test (instant refill) was eventually obtained in all of them, and there was a clinically unobstructed airway in the remaining 79 patients.

We conclude that a negative ODD test (instant refill) indicates that clear airway has been achieved, a positive test indicates misplacement of the LMA and an equivocal test indicates that the patient is too light. We consider that the ODD is useful in the placement of the LMA since it identifies both incorrect placement and inadequate depth of anaesthesia.

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Persistent erection and general anaesthesia

The letter from Drs Selby and Sugden (*Anaesthesia* 1989; **44**: 1016) prompts this report.

Most anaesthetists encounter this problem occasionally, typically during a cystoscopy on a fit, young man and, although I have encountered it in no more than two or three patients, I have used a small dose of ketamine with rapid and consistent success. Twenty to 30 mg intravenously is sufficient to produce detumescence within a minute or two, totally without unwanted effects. No emergence reactions have ensued and even a day patient was able to return home as planned.

This dose of ketamine is too small to produce reliable anaesthesia alone, but nevertheless it is prudent to recommend its use only where anaesthetic expertise is immediately available.

It is not too surprising that ketamine should have such an effect since its effects on blood pressure and heart rate are thought to be a sympathetically-mediated phenomena¹ and, in addition, the dissociative action may interfere with

the erectile process just as over indulgence in alcohol is renowned to do.

Whatever the rationale, this treatment is simple, free from arrhythmogenic effects and less traumatic than direct injection into the corpus. The drug has the additional advantage of being readily available in most anaesthetic rooms (whereas it may be much more difficult to obtain terbutaline, for example, without considerable delay).

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Reference

1. MARSHALL BE, WOLLMAN H. General anaesthetics, In: Goodman LS, Gilman AG, Rall TW, Murad F. *The pharmacological basis of therapeutics*, 7th edn. Philadelphia: Macmillan p. 298.

An inappropriate use of tape

A Boyle's machine and Manley Pulmovent MPP 2000 were checked before an operating list. The oxygen analyser was used when checking the Boyle's machine, but was not attached when the ventilator was tested.

Ventilation did not occur when the patient was connected. However, the lungs were easily ventilated manually (on the manual setting) and the arterial oxygen saturation remained within normal limits. The T-piece of the oxygen

analyser was then observed to be taped and a crack detected. Automatic ventilation was satisfactory when the T-piece was removed from the system.

The pressure developed at the common gas outlet (CGO) with a Manley Pulmovent MPP 2000 was measured. This was done by inserting a sphygmomanometer, attached to a T-piece, in series with the ventilator connexion at the CGO, where the pressure varied between 11 kPa and 14

kPa at flows of 2 to 20 litres/minute. The ventilator was found to require a pressure of 14 kPa to initiate inspiration. The crack, which extended beyond the tape on the T-piece of the oxygen analyser, produced a leak which prevented the pressure from rising above 11.5 kPa which was not adequate to trigger the ventilator.

It is clear that the reason for the tape on the equipment should be fully investigated before a list, since the use of tape is a notorious hazard.

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More on monitoring

Few monitoring devices currently offer moment-to-moment monitoring of ventilation, although of course transcutaneous sensors and expired gas analysis are invaluable guides to gas exchange, and several monitors also produce a waveform with each chest expansion. What we really need is some real-time device that will detect and signal minor, inaudible airway obstruction, accumulation of secretions in the airway, slight wheezing ('bronchospasm') and slight changes in the pattern of spontaneous breathing that may signify changes in the depth of general anaesthesia. Of course we all watch the bag, look at the pressure dial and spirometer of the ventilator and listen to the chest

casually with a stethoscope. Listening over the laryngeal area (or to the end of a T-piece or over the ventilator tubing) is invaluable, although this is not widely taught. Apparently there are major technical problems in producing a device that can not only link a throat microphone to a loudspeaker but exclude the considerable extraneous and artefactual noise with a filter. Maybe some boffin can work on the problem!

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Another failure of Selectatec block

The second patient for arthroscopy on a day-case list had previously received halothane, so isoflurane was to be used. The anaesthetic machine in both the anaesthetic room and the operating theatre had 'Selectatec' vaporizer fittings and shared a single isoflurane Mark III vaporizer.

Anaesthesia was induced with propofol afentanil, N₂O, O₂ and isoflurane and the patient was transferred from the anaesthetic room to the operating theatre after manual ventilation of the lungs. The operating department assistant preceded the transfer of the patient, removed the halothane Selectatec from the theatre Boyle's machine and substituted the isoflurane vaporizer. The reservoir bag would fill when manual ventilation was restarted with a Bain system, but a large leak occurred. Sufficient inflating pressure could not be maintained to ventilate the patient's lungs.

Rapid inspection of the machine and system indicated no cause for the leaks so the emergency O₂ flush was used to fill the system and ventilation was continued whilst a substitute Boyle's machine was produced. Careful examination subsequently revealed that on removing the halothane Selectatec, the 'O'-ring for the right-hand pin had been

removed inadvertently with the vaporizer. Thus a non gas-tight junction resulted when the isoflurane Selectatec vaporizer was attached.

The degree of leak resulted in a fresh gas flow sufficient to fill the Bain system in a spontaneously breathing patient, but was insufficiently gas tight when a large pressure was applied to the system, as in manual ventilation. Failure to 'seat' a Selectatec vaporizer is a well recognised complication. Selectatec switch malfunctions can also result in cessation of gas flow. No report of a lost O-ring has been received by the Department of Health. It is surprising that this event has not been reported previously since the ease of O-ring removal is evident on examining the Selectatec block.

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Reference

1. HOGAN TS. Selectatec switch malfunction. *Anaesthesia* 1985; **40**: 66-9.

Failure of a valve in a Bain system: a dangerous design

It has been pointed out to us that the last sentence in the letter from Dr Breen could be misinterpreted (*Anaesthesia* 1990; **45**: 417-8) and we overlooked this. Cory Bros would like to make it clear that the Bain valves in question were serviced and returned to the hospital within a few days and that the valves are in routine use and working perfectly

since that time; in fact, we understand that they are preferred by most of the users.

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Book reviews

Cardiopulmonary resuscitation
Edited by W. KAYE AND N.G. BIRCHER

796

Monitoring in anaesthesia and critical care medicine 796
Edited by C.D. BLITT
Books received 797

Cardiopulmonary resuscitation

Edited by W. KAYE AND N.G. BIRCHER. Pp. xi+231
Churchill Livingstone, 1989. £30.

Correctly addressed to its target audience this book is a specialist's tool mainly for those seeking to advance the discipline of resuscitation or wanting a reference text. Written largely by American authors it comes from the Critics in Critical Care Medicine series, and is not a basic manual despite its simple title. It is a collection of essays and its 11 chapters vary as widely in approach as in length, and deal with the theory, practice, appropriateness and outcome of cardiopulmonary resuscitation. If any special justification were needed to interest anaesthetists the sole UK contribution handles peri-operative resuscitation. References are legion: the first two chapters contain 294 and 470 respectively and their citation in the text in such numbers and the liberal use of abbreviations make some chapters easier to consult than to read. Illustrations include clear tables often summarising quoted studies, and photos of the doyens of the art. Only the chapter dealing with paediatric aspects had figures, and it was the better for them. The index was modest and I found browsing through the relevant chapter a better way of finding material.

The first quarter of the book is Peter Safar's historical treatise and its length must be justified by the need to learn from the past but I also simply enjoyed it. Physiology and pharmacology are dealt with rather succinctly in a chapter that serves well as source of other references. It was surprising that more room had not been found somewhere in the book for broader handling of many topics such as drowning, arrhythmias and pacing. Agreed it can be argued that the last two fall to a certain extent in the area of prolonged rather than basic or even advanced life support, and granted that other books in the series might cover such related topics, but had a restrictive policy been strictly applied, for example to the chapter on peri-operative resuscitation, it would have been much the poorer. As it is, it had a healthy pre-emptive air and its strength lay in its wider remit.

Outcome, firstly of the mechanics of resuscitation by 'citizens' and professionals and secondly of the teaching of skills are key chapters. For the first, the quotation of one study in which the average time between discovery of cardiac arrest in the street and arrival of paramedics of 2.1 minutes suggests a certain zeal not always so easily discernible in the UK. For the second, the chapter on teaching and its evaluation could be read as a general example for instituting any training scheme, and for all the minor criticisms likely of a text written by many authors, it is this chapter that is to me at the heart of the matter. If you need to establish a training programme buy this book. Trainees and those needing to improve their personal skills should look elsewhere.

J. CURRAN

Monitoring in anaesthesia and critical care medicine.

Edited by C.D. BLITT. Pp. xix+903. Churchill Livingstone, 1990. £75.

This is the second edition of a book that was very well received when first published in 1985. Its intended readership is inferred from the title and the topics covered include most of those relevant to anaesthetists in these two spheres of activity.

The plan of the book is logical: it starts off with basic principles and goes on to detailed descriptions of the various techniques and facilities available. An attempt is made to achieve comprehensive cover and up-to-date references in each of the sections, and with due allowance for the time taken for publication this has been achieved. Any review would have to be as long as the book if it were to be all embracing, but there are certain notable points which should be made.

There is a comprehensive review of cost benefit analysis of monitoring techniques after an introductory chapter and a good discussion of monitoring and patient safety. Some of these are based on entirely hypothetical assumptions, but they appear to make very good sense. The authors' computation of the cost of saving one life or of reducing hospital stay through the avoidance of errors by using monitoring equipment is quite revealing. The fact that the chapter ends with the statement that monitoring is clearly cost effective has to be taken in the context of spiralling medical costs.

There are then 250 pages concerned with various methods of cardiovascular monitoring. This begins with a most interesting review of noninvasive and invasive blood pressure monitoring, and the complications of intravascular monitoring are well covered. Swan Ganz catheterisation is discussed comprehensively, and a description is given of the many direct and indirect measurements that can be made with flow-directed catheters. The hazards and complications of the technique are covered in some detail and the danger of blind acceptance of the data provided is emphasised. It is in the main a very balanced section, although the inaccuracies and shortcomings of the method are mentioned rather too briefly.

Electrocardiography, including the use of transoesophageal ECG monitoring is discussed. Only a few of the anti-arrhythmic drugs available in the United Kingdom are described, but the whole section is clinically relevant.

The section on respiratory monitoring begins with a review by Dr Fairley that is a pleasure to read. It is followed by a section on monitoring anaesthetic and respiratory gases and a standard review of blood gas monitoring, and concludes with a short chapter concerned with pulse oximetry.

The second half of the book starts with a review of the methods available to monitor the central nervous system and neuromuscular junction. Methods of analysing the EEG are described in outline and anyone who wishes to

read further on the topic will find a very comprehensive list of references in this, as in all the other sections. There is a particularly good description of the value and limitations of intracranial pressure monitoring. The enthusiasm often expressed in this country for monitoring in such conditions as Reye's Syndrome is reviewed coolly.

Patient and equipment safety in the operating theatre are covered adequately and there is a chapter on computers in anaesthesia that again is very logical and helpful. Monitoring in neuroanaesthesia, cardiac anaesthesia, paediatrics and obstetrics bring up the rear, and after a surprisingly short chapter on the philosophy of monitoring in the intensive therapy unit, the book concludes with an attempt to predict future developments in monitoring.

Overall, this is an excellent book. It is beautifully produced, clear, easy to read and with many illustrations. Librarians assure me it is pointless to suggest that it is easier for libraries to buy books than individuals in which case I should end by suggesting that it should be read by as many people as possible who are attracted to the title.

J.C. STODDART

Books received

We thank the publishers for the following books, some of which may be reviewed in future issues of *Anaesthesia*.

Clinical respiratory physiology

A.E. TAYLOR, K. REHDER, R.E. HYATT AND J.C. PARKER. Pp. x+300. Saunders, 1990.

Drugs in anaesthesia and intensive care

M.P. SASADA AND S.P. SMITH. Pp. vi+266. Castle House Publications, 1990. £24.

Clinical applications of ventilatory support

Edited by R.B. KIRBY, M.J. BANNER AND J.B. DOWNS. Pp. xi+545. Churchill Livingstone, 1990. £49.95.

Anaesthesia review 7

Edited by L. KAUFMAN. Pp. x+243. Churchill Livingstone, 1990. £18.50.

Essentials of anesthesiology, 2nd edn.

D.C. CHUNG AND A.M. LAM. Pp. xiii+249. W.B. Saunders, 1990.

Scientific foundations of anaesthesia—the basis of intensive care, 4th edn.

Edited by C. SCURR, S. FELDMAN AND N. SONI. Pp. xiv+745. Heinemann Medical, 1990. £80.

Innovations in physiological anaesthesia and monitoring.

Edited by R. DROH AND R. SPINTGE. Pp. x+179. Springer-Verlag, 1989.

Correction

The Editor stated in a brief review of *The history of anaesthesia* (*Anaesthesia* 1989, 44: 1019) that there was no index in this book. This was incorrect.

Anaesthetic literature

This section of *Anaesthetic literature* contains references taken from *Current Contents—Life Sciences* for May 1990. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase II, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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Physiology

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Hazard Notices

Graseby Medical MS2000 Syringe Driver (HC Hazard 90:20). There have been a number of incidents in which MS2000 Syringe Drivers appeared to be operating satisfactorily but have in fact failed to infuse and alarm. These failures are related to loosened and inadequate screws that hold the gearbox and motor assembly. A simple repair is required.

Patient burns HC(Hazard)(90:25) warns again about the risk of pooling of spirit-based fluids which may be accidentally ignited during the use of surgical diathermy and result in burns to the patient.

THIS PAGE CONCERNS YOU IF YOU ARE
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PROFESSION WHO PRACTISES IN ANAESTHESIA
AND YOU WISH TO APPLY FOR ASSOCIATE MEMBERSHIP

Ordinary Membership (*United Kingdom and Ireland only*)

Voting membership of the Association (the only effective body representing anaesthetists' interests in non-academic matters).

The right of attendance at the Annual General and Scientific Meetings and all other meetings of the Association of Anaesthetists.

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Fill in the application form on the other side of this page.

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THE ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

APPLICATION FOR MEMBERSHIP

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The Association of Anaesthetists of Great Britain and Ireland
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(This application will be considered by Council at its next meeting. NO MONEY should be sent in the meantime. After acceptance of the application by Council, the Hon. Secretary will notify the applicant of Council's decision and the amount of subscription payable in the current financial year.)

.....19

To the Honorary Secretary,
Association of Anaesthetists of Great Britain and Ireland

I (full name) offer my name as a candidate

BLOCK LETTERS

for (a) ORDINARY MEMBERSHIP (b) OVERSEAS MEMBERSHIP (c) ASSOCIATE MEMBERSHIP (d) JUNIOR MEMBERSHIP of the Association *(please delete categories not applicable)**

I hold the following appointment(s) as an anaesthetist *(please state grade if in the National Health Service, and indicate the approximate proportion of time spent in the practice of anaesthesia):*

.....
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Qualifications (with dates).....

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(Signed).....

Address.....

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We, being Members† of the Association, nominate as a candidate for ORDINARY MEMBERSHIP/OVERSEAS/ASSOCIATE/JUNIOR MEMBERSHIP

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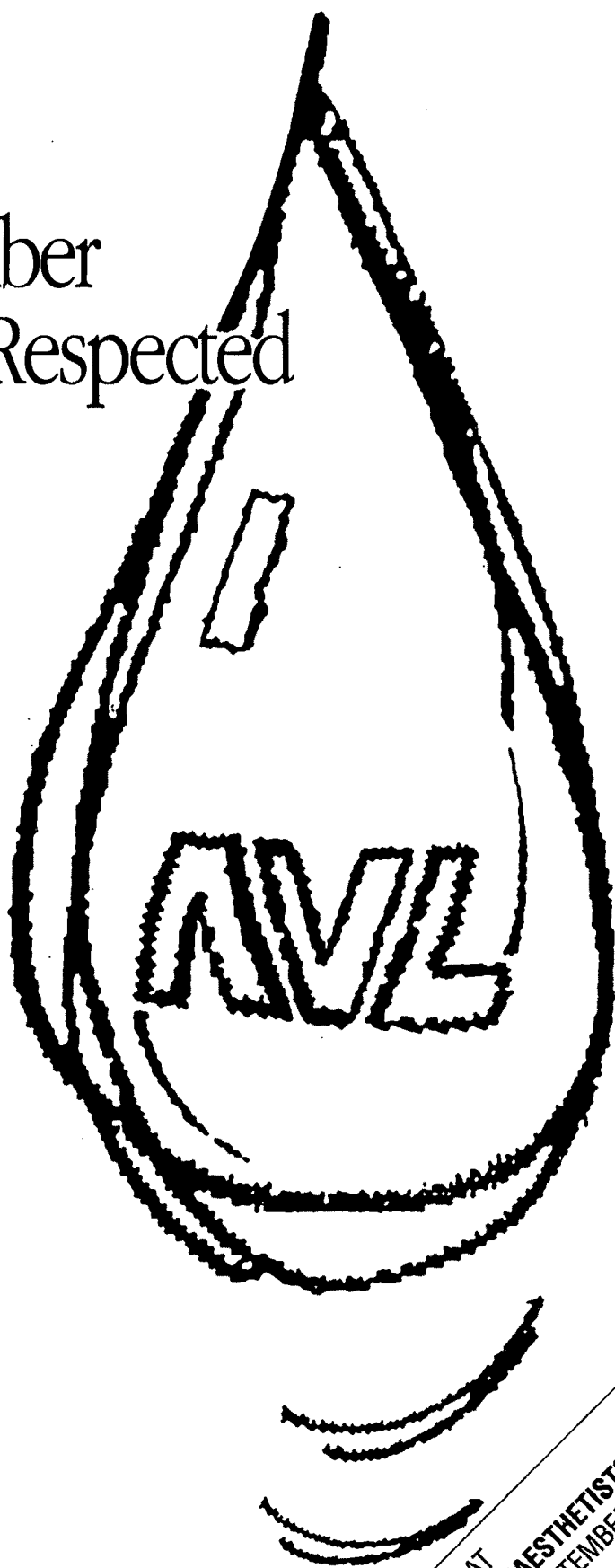
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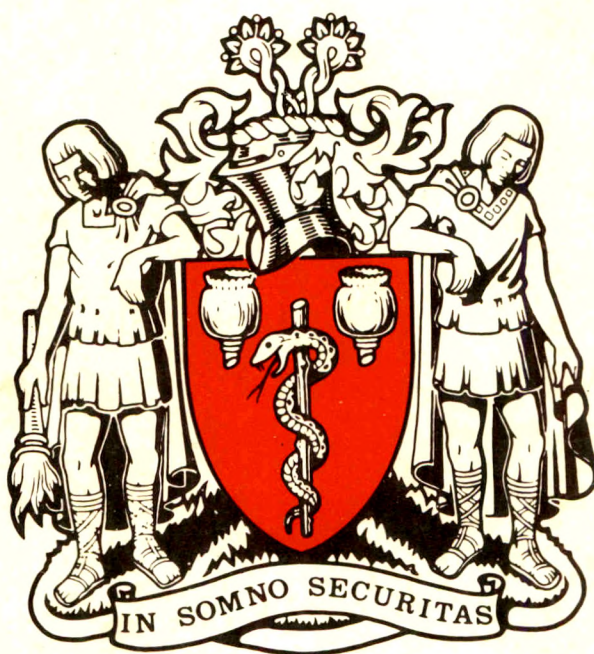
31 OCT 1990



Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 45 Number 10 October 1990



VCM



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Published for the Association by
Academic Press Grune & Stratton
London San Diego New York Boston
Sydney Tokyo Toronto



ISSN 0003-2409

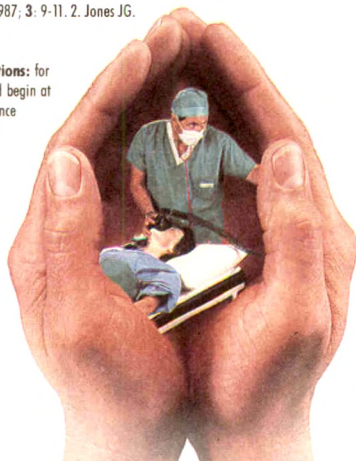
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ANAESTHESIA: ISSN 0003-2409, Volume 45 1990, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

All advertising enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293).

Annual subscription price including postage: £118 UK and US \$228 overseas. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel. 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

Anaesthesia is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

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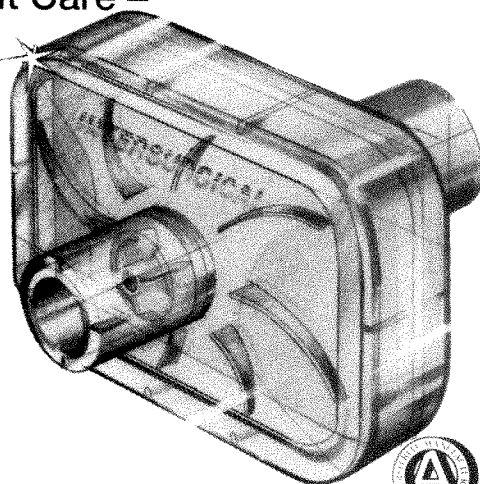
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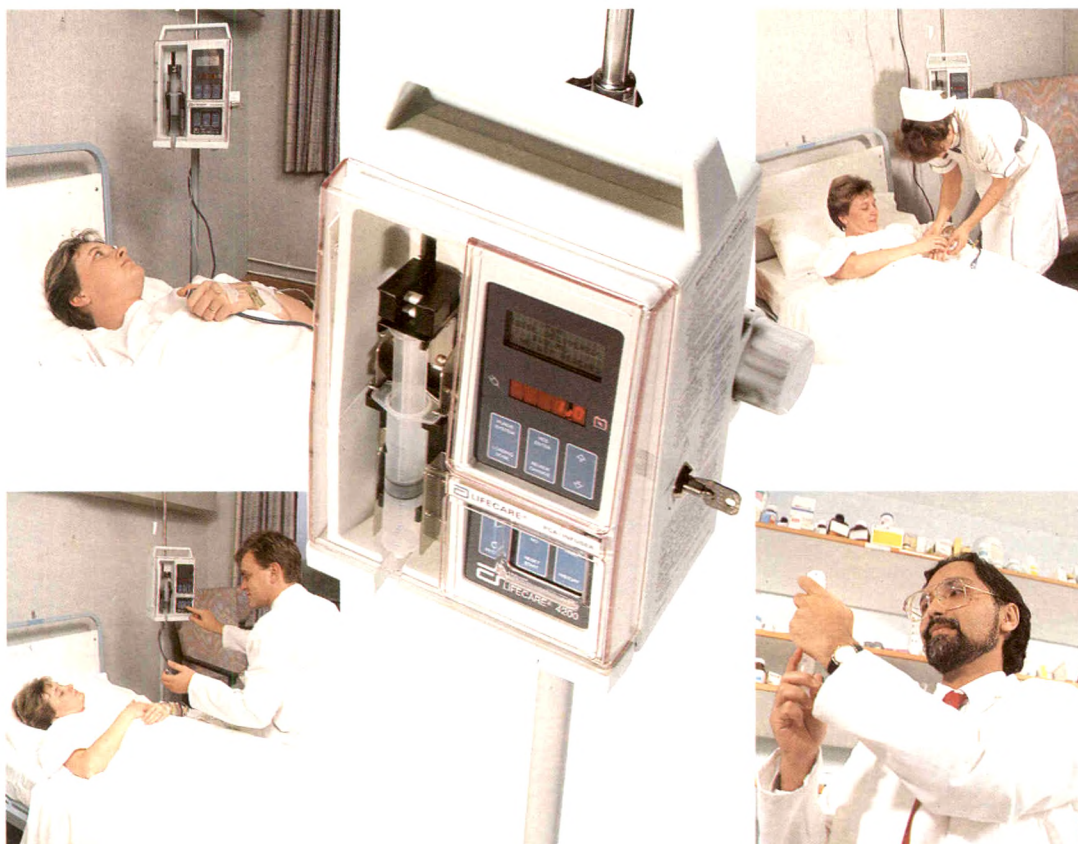
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Editorial

Anaesthetists and trauma

Injury is the commonest cause of death in both males and females between the ages of 1 and 35 years in the United Kingdom, and road traffic accidents head the list of causes. More than 4600 died on the roads in 1988: over 90 per week, every week;¹ a number that exceeds the carnage at Hillsborough Football Ground on that fateful Saturday in May last year. Even more staggering is the number of seriously injured, over 53 000 in 1988.¹ Each of these patients stayed 10-12 days in hospital; this accounts for approximately 700 000 bed days that year. There are more than two victims with permanent disability for every death on the roads, but almost as many die from injury in the home, although these incidents occur in different age groups, infants and the elderly. Industrial accidents account for a further 26 000 hospital admissions annually.²

These numbers do not include the headline-hitting incidents, such as rail and air crashes, terrorist explosions and hand-gun injuries which, even in Northern Ireland, account for only a small proportion of the total numbers of dead and injured. Forty-nine individuals died in 1984 in the UK from gunshot wounds, many of which were accidental. By contrast, in 1982 in the USA, there were 11 000 murders and a further 10 000 accidental deaths or suicides involving hand-guns.

Road accidents and industrial injuries involve the young and middle aged, and the ensuing socio-economic effects are particularly disturbing. There is, in this productive age group, the enormous cost of treatment, rehabilitation and welfare support of the dependants (in most cases borne by the State), together with a substantial loss of expertise in the work place and reduction in tax revenue to compound the economic disaster.

The Department of Transport in 1988 calculated the average cost of a single fatality from a road accident to be more than £550 000.¹ Thus the total figure for the 4600 plus who die on our roads amounts to over £2.5 billion. If the costs of fatal injuries at work, in the home, and from murder and manslaughter, and of nonfatal injuries are aggregated, the figure becomes quite staggering, over 1% gross national product.

The price of trauma cannot only be measured in terms of cash but also in talent lost to the nation, and in physical and psychological debility in survivors and their relatives and loved ones.

It was appreciated since before the inception of the National Health Service in 1948, that the management of trauma in the UK is, to put it kindly, suboptimal. A series of reports by the British Orthopaedic Association (BOA), The British Medical Association, The Ministry of Health and the Joint Consultants' Committee between 1943 and 1978 drew attention to the shortcomings in terms of facilities and, more particularly, the 24-hour availability of trained and experienced physicians in hospitals accepting accident and emergency patients.

Reports from other countries over the past 10-15 years have clearly indicated that a considerably greater

proportion of health care resources is applied to help trauma victims. Prehospital care in the United States is provided by highly trained paramedics, a development from experience gained in the war in Vietnam, which is also followed in Australia and South Africa. Emphasis is placed in Europe, on a physician-based service which has achieved remarkably high standards in the Federal Republic of Germany, in France (the SAMU and SMUR systems), in Belgium and in many of the Eastern European countries, notably Czechoslovakia and Hungary.

The Confidential Enquiry into Perioperative Deaths (CEPOD) published in 1987³ emphasised deficiencies in the National Health Service because it showed that a significant number of victims of trauma died in hospital partly because of the avoidable lack of resources and, importantly, because management of the seriously injured was left to relatively inexperienced junior medical staff without senior staff involvement.

Britain's trauma service is also criticised by overseas colleagues. Trunkey, from the United States, described the system he saw on a visit, as 'disorganised, fragmented and producing a universally bad outcome'. Trunkey is a surgeon who is respected worldwide, because he and his colleagues in San Francisco have clearly demonstrated the value of a single centre which specialises in trauma care compared with smaller emergency departments similar to our District Hospitals. His well planned study⁴ showed that only 1% deaths from trauma were judged to be preventable in the trauma centre compared with between 28% and 73% in ordinary hospitals. Many other studies supported these findings and demonstrated the impact of trauma centres on preventable death. The concept has been further developed throughout the USA by the initiative of the American College of Surgeons' Committee on Trauma.

Stimulated by this evidence and by the justified criticism of the service in the UK, the Royal College of Surgeons of England set up, through its Commission on the Provision of Surgical Services, a Working Party under the chairmanship of Professor M. Irving to assess 'The Management of Patients with Major Injuries'.⁵

The Working Party undertook both retrospective and prospective studies, which confirmed the findings of CEPOD and the criticism by Trunkey. Four out of four assessors in the retrospective study agreed that 20% (1000) patients who arrived in hospital alive with major injuries would be alive had they been managed in an American-style trauma centre.

The medical profession has responded to these reports by supporting the concept of concentration of patients with major injuries in centres with comprehensive specialist facilities and experienced senior medical staff always available. Doubts are expressed by some of those who work in hospitals which are unlikely to become designated trauma centres. These, predictable, opinions may be based on fears of loss of a substantial proportion of current workload and that the hospitals

may appear to be second-rate. However, major injuries comprise less than 5% of an average Accident and Emergency (A&E) Department's workload and referrals to specialist centres happen now. The problem in the UK is that few major centres can offer total trauma care; neurosurgery may be available in one centre, maxillofacial and plastic surgery in another and cardiothoracic surgery in yet another. The trauma centre aims to provide the patient with major injuries with the expertise of several disciplines. This will require a re-organisation and relocation of specialist facilities in many cities.

Implementation of an effective trauma service will not just require extra facilities in designated centres but will also need much re-education. This will include improved assessment on site by paramedical staff (Revised Trauma Scores) and better arrangements for stabilisation and rapid referral. Helicopter transfer between hospitals has an undoubted value and the benefits are known from experience in London, Cornwall, Wiltshire and Bristol. Costs are high, but so are the benefits.

There is considerable interest in the provision of a good trauma service, particularly among anaesthetists. The Advanced Trauma Life Support (ATLS) Courses, run under the auspices of the Royal College of Surgeons of England, are extremely popular and oversubscribed. Anaesthetists represent a substantial number of the instructors and candidates at each Course. Our Association has given several grants to anaesthetists who seek specialist trauma experience in the United States and a number of centres have established linked posts for those wishing to work in trauma centres for a year so that they can return to the UK with the benefit of their experience. Not all the American methods are applicable in the UK but we have a lot to learn from them about the concept of the trauma team approach. Similarly, it appears that we have something to teach in the United States, particularly about analgesia for the injured.

The Association has also established a Joint Working Party with the College of Anaesthetists and individual Consultants in A&E to clarify our role in the Emergency Service. This role includes involvement in training and practice in prehospital care, as well as resuscitation and anaesthetic services to the A&E Department. The College of Anaesthetists plans to arrange meetings for those training ambulancemen to paramedic standard.

The Department of Health, after consultation and some 18 months after publication of the Royal College of Surgeons' report, has agreed to upgrade one major A&E Department in England to trauma centre standard to assess the impact of such a service. This is a start but more centres are needed to provide a prompt nationwide evaluation in the face of what seems to be overwhelming evidence.

One improvement in attitude could be made right away in large District General Hospitals. There needs to be an increased sense of urgency by trainees in calling for help and an increased speed of response by senior medical staff. The response should be at a senior level, Senior Registrar or Consultant, not by a registrar or SHO with only a few months' experience.^{3,6} It is this practice which creates unforgivable delay and causes avoidable death and disability.

Managers at National, Regional and District levels must appreciate the current carnage caused by trauma

and recognise the potential value of improved treatment. They must be prepared to realise the value of centralisation of major trauma care from a number of Districts and create sub Regional Centres for specialised care. Priorities should not be influenced by petty considerations of financial cross-boundary flow currently prevalent as a result of The White Paper proposals.

A fully staffed operating theatre should be available 24 hours a day, set aside purely for the treatment of emergencies. Urgently needed surgery should not be delayed by lack of operating room space because all the facilities are in use for routine lists.^{3,6,7} The object of having managers in the National Health Service is to create and expedite opportunities for better patient care, not to delay or obstruct such opportunities. Clear-cut cases of need and potential benefit require rapid and incisive decisions: the hallmark of good management in the commercial world. Paper and committees, and delays, cost a great deal in terms of both money and time and, in this instance, lives. A case of this urgency should not have to be remade again and again at quarterly or six-monthly intervals.

The cost of better trauma care will be high. Trauma centres will probably cost about £2-3 million each to add onto a suitable hospital. The national capital bill would be about £80 million. Staff and maintenance costs would probably be £1-2 million each, £50 million, nationally.

If a thousand lives could be saved each year, then £550 million could be conserved every year. Balanced against the costs above, this has to be one of the best investments ever in cash terms alone. It ignores the 1000 patients, and their families, who also will be grateful. Is it too much to ask that the government takes an overall look at its waistcoat pockets? Transferring some of the money from the one with a huge hole in it could result in enormous savings for the nation overall.

P. J. F. BASKETT
President

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7. Association of Anaesthetists of Great Britain and Ireland and The Association of Surgeons of Great Britain and Ireland, 1989. Report of the Working Party on Efficiency of Theatre Services.

Editorial notices

Observant readers will note that we have not formally welcomed Dr D.E. Newton (*Northwick Park*) as Assistant Editor for the last 17 months. We apologise to him for this unintentional slight. Readers will also note the appointment of Dr R.A. Mason (*Swansea*) as Assistant Editor. We welcome both of them and look forward to their continued contribution to the journal.

The Annual General Meeting of the Association of Anaesthetists of Gt Britain and Ireland is expected to confirm the appointment of Dr M. Morgan (Associate Editor) as Editor with effect from 1 October 1990.

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; 1: 432 5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Quality of life after intensive care

S. A. RIDLEY AND P. G. M. WALLACE

Summary

An important aspect of effectiveness of intensive care services is change in the quality of life of survivors after critical illness. A questionnaire was compiled using established methods for assessment of quality of life and sent to all known survivors of a regional intensive care unit. Each patient's quality of life was then quantified using disability categories. The results show that patients with a good premorbid quality of life suffered a significant decline after critical illness. Similar important decreases in quality of life were found in younger patients and trauma victims. Quality of life may be a valuable consideration in determining the appropriateness of intensive care management.

Key words

Intensive care; quality of life.

Effectiveness and outcome of intensive therapy services in the United Kingdom have been measured previously in terms of mortality.^{1–5} Mortality alone may be regarded as a relatively crude estimate of effectiveness if quality of life (QOL) enjoyed by the survivors after treatment is ignored.⁶ Previous studies have been performed outside the United Kingdom to investigate QOL after discharge from intensive therapy units (ITU) but have measured QOL in descriptive terms or concentrated on one aspect of health status.^{7–10} Little work has been undertaken to examine the QOL of patients discharged from ITU in the United Kingdom despite a recognised need.¹¹ The aims of this study were to quantify QOL before and after ITU admission and to relate any changes in QOL to age, diagnosis and severity of illness.

Methods

The study was approved by the hospital's Ethics Committee. All patients who were discharged from the ITU at the Western Infirmary, Glasgow between June 1985 and July 1987 were studied. The patients' ages and diagnoses were recorded and their severity of illness at admission was calculated using the APACHE II scoring system.¹² Unnecessary distress to relatives of patients who had died after discharge was avoided by communicating details of all ITU survivors to the Registrar General for Scotland, who identified patients who had died since discharge and provided copies of each death certificate.

A self-completed questionnaire was compiled from

previously validated methods to assess each patient's QOL before and after ITU admission (appendix A). This was based predominantly on a questionnaire described by Williams¹³ to measure QOL using Rosser's Disability Categories.¹⁴ However the questionnaire also included measures of functional capacity as described by Goldman,¹⁵ Patrick's perceived quality of life score¹⁶ and Katz's activities of daily living.¹⁷ The patient's QOL was calculated using the assignment rules described in detail in Appendix B. Scores were calculated from the perceived quality of life responses to assess the patient's usual activities, and social and personal relationships.

A questionnaire together with a stamped addressed envelope was sent to each known survivor using addresses recorded in the ITU admission log. The registration office of the Primary Care Departments at the local Health Boards were asked for more recent addresses if questionnaires were not returned or were returned undelivered by the Postal Service, and a second questionnaire was sent. A third attempt was made to contact the patients if no response was forthcoming.

The Wilcoxon signed rank test was used to determine the significance of changes in categories of QOL. A *p* value of less than 0.05 was considered significant.

Results

During the 2-year period, 385 patients were discharged from the ITU at the Western Infirmary and at the time of this study 129 of these were identified by the Registrar

S.A. Ridley, FFARCS, Senior Registrar, P.G.M. Wallace, FFARCS, Consultant, Division of Anaesthesia, Western Infirmary, Glasgow G11 6NT.

Accepted 28 February 1990.

Table 1. The distribution of the diagnostic categories, age and severity of illness (median values with 10th and 90th centiles given in parentheses).

Diagnostic categories	Number	Age (years)	APACHE score
Neurological	3	49 (37-52)	7 (2-24)
Cardiovascular			
sepsis	17	44 (30-67)	10 (5-15)
hypovolaemia	11	65 (35-80)	14 (2-20)
bleeding	10	55 (17-62)	11 (2-28)
Gastrointestinal obstruction/perforation	9	60 (32-72)	9 (7-22)
Trauma	23	20 (17-69)	8 (2-17)
Respiratory			
postoperative	46	50 (20-72)	9 (2-21)
failure	19	60 (18-77)	12 (7-23)
Poisoning	11	30 (19-68)	16 (1-23)
Postcardiorespiratory arrest	4	59 (18-83)	10 (2-15)
Miscellaneous (including renal failure)	32	34 (21-71)	11 (5-24)

Table 2. Distribution of changes in disability category after treatment in ITU.

Disability category	Change in category											
	Improvement				No Change				Deterioration			
	n	+4	+3	+2	+1	0	-1	-2	-3	-4	-5	-6
I (Good)	29					15	5	1	0	3	3	2
II	31				2	19	8	1	0	0	1	
III	38			3	8	18	3	2	2	2		
IV	6		1	1	0	3	0	1				
V	11	1	1	1	0	6	2					
VI	6	1	1	0	2	2						
VII (Poor)	5	1	0	0	0	4						

Disability categories for 30 patients were not calculated because of missing replies either before or after ITU.

Table 3a. Distribution of employment groups and change after critical illness.

Employment	Change in employment groups											
	Improvement				No change				Deterioration			
	n	+5	+4	+3	+2	+1	0	-1	-2	-3	-4	-5
Categories/groups												
1 Disabled	17	1					16					
2 Retired	34						28	6				
3 Housewife	18						18					
4 Unemployed	19				2	2	14	1				
5 Part time employment	9					3	5	1				
6 Full time employment	39						30	0	2	1	2	4

(* = $p < 0.05$. Twenty patients did not complete employment section fully).

Table 3b. Distribution of APACHE scores and changes in disability category after ITU.

APACHE score	Change in category											
	Improvement				No change				Deterioration			
	n	+5	+4	+3	+2	+1	0	-1	-2	-3	-4	-5
0-9	52		3	2	2	4	27	6	3	0	4	1
10-19	61			1	3	8	31	10	3	0	1	2
>20	13				1	0	7	3	0	1	0	1

Table 4a. Distribution of age and changes in disability category after ITU.

Age; years	n	Change in category											
		Improvement			No change				Deterioration				
		+5	+4	+3	+2	+1	0	-1	-2	-3	-4	-5	-6
<30	29			1	2	1	15	7	1	0	2		*
30-59	57		2	1	3	6	31	6	4	1	1	2	
>60	40		1	1	1	5	19	6	1	0	2	2	2.

Table 4b. Distribution of diagnostic categories and changes in disability category after ITU.

Diagnosis	n	Change in categories											
		Improvement			No change				Deterioration				
		+5	+4	+3	+2	+1	0	-1	-2	-3	-4	-5	-6
Cardiovascular (including sepsis)	20					2	11	1	3	0	0	2	1
Respiratory	41		2	2	2	4	21	4	2	1	2	0	1
Gastrointestinal	23		1	1	2	4	10	3	0	1	1		
Trauma	26				1	0	13	8	1	0	2	1	*
Miscellaneous	16				1	2	10	3					

(* = $p < 0.05$).

General as having died. The remaining 256 patients were each sent a questionnaire. Seventy-one questionnaires were ultimately returned undelivered by the Postal Service after three attempts. A total of 185 patients received the questionnaire, 156 were returned completed and 29 were not returned, or were returned not completed (response rate 84%).

The age, APACHE score and diagnostic categories of the 185 surviving patients are illustrated in Table 1.

The changes in QOL before and after ITU are described in Table 2. Patients admitted with a good QOL (i.e. Disability Categories I or II) suffered a statistically significant decrease in QOL after critical illness ($p < 0.05$) while those with a lower pre-admission QOL experienced no significant change. Table 3 shows the distribution of severity of illness and changes in employment group after critical illness. Patients who had previously been fully employed suffered a significant decrease in their employment category ($p < 0.05$). Furthermore, significant decreases in QOL were observed in younger patients (aged < 30 years) and in trauma victims ($p < 0.05$) (Table 4). Other age groups or diagnostic categories showed no significant changes.

Discussion

The assessment of QOL is difficult and numerous techniques have been described. At present the best validated indices of health are the Sickness Impact Profile,¹⁸ the Quality of Well-Being Scale,¹⁹ the McMaster Health Index Questionnaire²⁰ and the Rosser Disability Categories. The present questionnaire, although not validated formally, concentrated on Rosser's Disability Categories which are well known in this country and based on a British population. It also forms the basis for the calculation of 'Quality Adjusted Life Years' (QALY). Patrick's 'Perceived Quality of Life' score was included because of its relative simplicity. Some repeated question themes had to be omitted in order to avoid repetition in a questionnaire which encompassed four QOL measures, and therefore transformation of some replies became essential. Transformation of the Perceived

QOL scores to Rosser's Disability Categories can be undertaken legitimately,¹³ but the generated data are not strictly comparable with those from other studies.

Patients in the present study had been discharged 1 to 3 years before questioning and this may have affected memories of QOL before ITU. A more detailed QOL measure could have been used if survivors had been interviewed in person. Despite these limitations, this questionnaire has detected changes. The results indicate that many patients had a poor QOL before admission to ITU, but there was an important decline in QOL after critical illness in those who had previously enjoyed a good QOL. It is particularly interesting that young patients and trauma victims (patients who may benefit the most from ITU management in terms of survival) exhibited significant decreases in their QOL. In these subgroups, it is likely that the long-term effects of serious orthopaedic injuries account for the decreases in QOL. Interpretation of the changes in employment groups for those who were previously fully employed is difficult because other factors such as local economic considerations may be more important than the critical illness itself.

These results conflict with reports from outside the United Kingdom which have detected no significant change in QOL after critical illness.^{8,9} In addition, Jacobs *et al.*⁸ in the Netherlands proposed that the best indicator for QOL after ITU was health status before the acute illness. The present study suggests that this may not be the case in the United Kingdom. The differences may reflect dissimilar groups of patients and ITU facilities compared to those in continental Europe.

Our results suggest that a more comprehensive evaluation of the effect of critical illness on QOL should be undertaken.

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Appendix A

Appendix A: self-completed questionnaire

Please tick or mark at the appropriate place the answer which best describes what you were able to do *before* your admission to intensive care ward (ITU) and what you are able to do *at present*.

1. Activity levels

Which level of activity describes your abilities? Please tick the most appropriate answers:

	(a) before admission to ITU	(b) at present
1. Disabled	—	—
2. Retired	—	—
3. Housewife	—	—
4. Unemployed	—	—
5. Part-time employment	—	—
6. Fully employed	—	—

2. General mobility and activities

Which of these statements best describes your abilities? Please tick one of the answers in each column.

	(a) before admission to ITU	(b) at present
1. I am not disabled in any way.	—	—
2. I am limited in some recreational activities (e.g. unable to continue a hobby such as playing football) but I can move around indoors and outdoors on my own.	—	—
3. My performance at work is slightly impaired or as a housewife I am able to do all housework except very heavy tasks.	—	—
4. What I can achieve at work and at home is severely limited.	—	—
5. I can get about indoors and outdoors on my own but I have to use a walking aid (e.g. stick, frame, crutch, wheelchair, artificial limb).	—	—
6. I am unable to undertake any paid employment or do any housework.	—	—
7. I am confined to the house, needing someone's help to get outside.	—	—
8. I have to spend nearly all my time in bed.	—	—

3. Perceived quality of life

How satisfied are you with the following aspects of your life? Please score on a scale of 0 to 10 (0 = not satisfied at all, 10 = completely satisfied).

	(a) before your admission to ITU score:—	(b) now score:—
1. The state of your health.	score:—	score:—
2. Your ability to think and remember.	score:—	score:—
3. How happy you are.	score:—	score:—

4. How much you see your family and friends. score:— score:—
5. The help you get from family and friends. score:— score:—
6. Your contribution to the community. score:— score:—
7. Your activities outside work. score:— score:—
8. How your income meets your needs. score:— score:—
9. How respected you are by others. score:— score:—
10. The meaning and purpose of your life. score:— score:—
11. With working/not working/retirement. score:— score:—

4. Distress

How much does your state of health distress or worry you overall? Please score on a scale of 0 to 10 (0=*not* distressed about my state of health at all, and 10=*very* distressed indeed about my health) (a) before admission to intensive care ward —; (b) at present —.

Please complete the following section only if you are disabled.

5. Activities of daily living

Please answer YES or NO to the following questions about the activities of daily living (please circle Yes or No as appropriate).

- | | (a)
before
your
admission
to ITU | (b)
now |
|---|--|------------|
| 1. Bathing | | |
| – Do you use any equipment to help you bathe (e.g. hand rails)? | Yes/No | Yes/No |
| – Does anyone help you with any equipment, supervise you or bathe more than one part of you? | Yes/No | Yes/No |
| 2. Dressing | | |
| – Do you use any special clothing (zipperpulls, velcrofasteners)? | Yes/No | Yes/No |
| – Except for tying your shoes does anyone help you get dressed, supervise your dressing or get your clothes from closets or drawers? | Yes/No | Yes/No |
| 3. Toileting | | |
| – Do you use a raised toilet seat, or hand rails to lower or raise yourself from the toilet? | Yes/No | Yes/No |
| – Do you use a bed pan or commode during the day or does anyone help you to and from the bathroom, clean you or adjust your clothing? | Yes/No | Yes/No |

4. Movement
 - Do you use a cane or a walker or other equipment to help you get in and out of chairs and your bed? Yes/No Yes/No
5. Continence
 - Do you ever take medicine or enemas to control your bowels? Yes/No Yes/No
 - Do you have another person help by giving you enemas, a catheter or bed pan, or do you have occasional accidents? Yes/No Yes/No
6. Feeding
 - Do you have special dishes or cutlery or does anyone butter your bread or cut your meat? Yes/No Yes/No
 - Does anyone ever feed you your meals or are you ever fed by tube? Yes/No Yes/No

Appendix B

Conversion of questionnaire responses to disability categories

Disability

General mobility (GM). General mobility replies scored according to the lowest level of mobility. Positive replies to questions 1 or 2 give GM score 1. Positive responses to question 3 give GM score 2; positive replies to question 4 give GM score 3 and so on until positive replies to questions 7 and 8 give GM score 6.

Usual activities (UA). Usual activity level score is determined by the sum of the scores for questions 1, 2 and 11 in the perceived quality of life section according to the assignment rules below:

Sum of replies	UA code
<7	4
8 to 15	3
16 to 22	2
23 to 30	1

(for missing replies, the UA code limits were scaled down, i.e. if only two completed, the sum of replies dropped to 20 and the code limits changed to <5, 5 to 10, 10 to 15 and >15).

Social and personal relationships (SP). Social and personal relationships score is calculated by the sum of the scores for questions 4, 6, 7 and 10 in the perceived quality of life section according to the assignment rules below:

Sum of replies	SP code
<8	4
8 to 16	3
17 to 24	2
25 to 32	1
33 to 40	0

(missing replies dealt with as above)

Self care (SC). Each 'yes' response to a separate question in the disability section scores 1 up to a maximum score of 4.

Assignment rules (after Williams)¹³

Disability. The table below indicates the disability category according to the patient's codes for GM, UA, SP, and SC. For the first five rows the score codes for SP and SC will be relevant.

Other responses	General mobility responses					
	1	2	3	4	5	6
UA=1						
SC=0 and SP=0	I	II	III	V	VI	VII
UA=1						
SC=1 or 2 or SP=1 or 2	II	II	III	V	VI	VII

UA=1						
SC=3 or 4 or SP=3 or 4	III	III	IV	V	VI	VII
UA=2 but						
SC<3 and SP<3	III	III	III	V	VI	VII
UA=2						
SC>3 or SP>3	III	III	IV	V	VI	VII
UA=3	IV	IV	IV	V	VI	VII
UA=4	V	V	V	V	VI	VII

Admissions to the intensive care unit after complications of anaesthetic techniques over 10 years

2. The second 5 years

J. M. LEIGH AND J. A. TYTLER

Summary

Compared with the first 5 years there was a 19% increase in general anaesthetics, a 171% increase in local and (or) sedation techniques and a 9% increase in obstetric epidurals with no increase in anaesthetic staffing. In this second 5-year period, 46 patients were admitted to the Intensive Care Unit as a result of a complication of an anaesthetic technique. These patients represented 1 in 2371 anaesthetic techniques carried out in the District compared with the previous 5 years where the incidence was 1 in 1543. Seven patients died (15.2%). The complication was considered to be wholly or partially avoidable in 14 instances (30.4%). Four of these subjects died.

Key words

*Anaesthesia; audit.
Intensive care.*

The 5 years reported here represents the second half of a 10-year prospective study period.

We have studied all the patients admitted to the Intensive Care Unit (ICU) in our District whose main admission diagnosis was encoded as 'Anaesthetic Complication'. The purpose was to establish the range and incidence of different complications, to identify predisposing factors if any, and to comment on the avoidance of such incidents with a view to improving practice.

The reason for the two separate reports of the 5-year periods is that we are aware of a large increase in workload, without any change in the establishment of anaesthetic staff, and wish to assess the impact of this change on the incidence of anaesthetic complications or its offset as a result of more knowledge, better drugs, techniques or monitoring equipment.

Methods

Details of all patients admitted to the ICU at the Royal Surrey County Hospital from 1 January 1984 to 31 December 1988 with an admission diagnosis of 'Anaesthetic Complication' were collected prospectively. These were patients for whom admission to the ICU was not planned before the operation. We again used the definition of a complication of anaesthesia as 'an undesirable effect attributable to a practical procedure or drug used for

analgesia or sedation or as part of an anaesthetic technique, not necessarily involving an anaesthetist'.

The hospitals in South West Surrey District provide services for all specialties except cardiac surgery and neurosurgery. The operating registers of all hospitals were collated so that the numbers and different types of anaesthesia or analgesia techniques carried out in the 5-year period were determined.

Results

Both the workload figures (Table 1) and admissions to the ICU have increased with a main admission diagnosis of 'anaesthetic complication' in 1% of ICU admissions (last 5-year period, 2%). The overall rate of complications which necessitated admission to ICU was 46/109 060 (53/81 780) or 1 in 2371 (1 in 1543).

The workload figures for the two 5-year periods show a one third increase. However, they conceal the steady rate of increase. There is a 91% increase in the population 'at risk' from anaesthetic techniques (from 12 682 to 24 171) when the first year is compared with the 10th year of the study period. The main increase was 497% in local and (or) sedation techniques (from 921 to 5497); the increase in general anaesthetics was 60% (from 11 134 to 17 795); and there was a 40% increase in obstetric epidurals (from 627

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Accepted 15 January 1990.

Table 1.

Year	ICU admissions	General anaesthesia	Local and (or) sedation	Obstetric epidurals	Total	Complications	Incidence
1979	251	11 134	921	627	12 682	7	1812
1980	473	13 240	1076	613	14 929	13	1148
1981	469	15 036	1636	645	17 317	15	1154
1982	643	15 587	1820	596	18 003	8	2250
1983	815	16 003	2227	619	18 849	10	1885
Total	2651	71 000	7680	3100	81 780	53	1543
1984	803	16 698	3170	514	20 382	9	2265
1985	862	16 971	3550	528	21 049	3	7016
1986	857	16 784	3986	704	21 474	8	2684
1987	889	16 587	4650	747	21 984	12	1832
1988	948	17 795	5497	879	24 171	14	1727
Total	4359	84 835	20 853	3372	109 060	46	2371
Total	7010	155 835	28 533	6472	190 840	99	1928

to 879). There has been no increase in anaesthetic staffing during the 10 years.

Time of identification of complications

The majority of complications occurred in the recovery period as before. (Fig. 1). The most frequent single cause was inadequate ventilation after reversal of muscle relaxants; 20 cases. The causes of complications that occurred during induction, maintenance and recovery are summarised in Tables 2–4. No complications occurred after local or regional anaesthesia.

Gender and age

There were 21 males with an average age 62 (SD 25, range 2–82) years and 25 females, average age 56 (SD 27, range 7–88) years. The entire group had an average age of 59 (SD 25) years. These data are very similar to those of the first 5 years.

Length of stay in ICU

The range of stay varied from 2–419 hours, but the patients could be divided into two subgroups on the basis of duration of stay. Forty-one patients each spent less than 75 hours in ICU and occupied 46% of the total time (845 hours: mean time in ICU 21, SD 13 hours; SEM 2 hours; range 2–55 hours). The five patients, who spent more than 75 hours each, occupied 54% of the total time (1000

hours—mean time in ICU 200, SD 133; SEM 54 hours; range 96–419 hours).

The mean age of the patients who stayed less than 75 hours in ICU was 56, SD 25 years and the mean age of the patients who remained in the Unit for more than 75 hours was 81, SD 37 years. The death rate in the group who stayed less than 75 hours was 5/41 (12%) and the death rate in the other group was 2/5 (40%).

Emergencies

Eleven patients were admitted after emergency operations and 35 after elective procedures. The corresponding figures for the previous 5-year period were 27 and 26 respectively. Nine thousand seven hundred and forty emergency operations were performed during the 5 years. Thus the risk of ICU admission was 1 in 885 for emergency, and 1 in 2837 for elective cases; this is slightly greater than a threefold difference. These figures represent improvements over those in the first 5 years; particularly after emergency procedures, from 1 in 388 and after elective cases from 1 in 2655 (7% better).

Deaths this 5 years (last 5 years)

Six patients died in ICU (8) and one within 24 hours of leaving it (1); this is an overall mortality rate within the group of 15% (17%). Four of these (5) were considered partially or totally attributable to the anaesthetic complication, and in the other three who died (4) the primary pathology and surgical factors contributed mainly to their deaths.

Figure 1 demonstrates the relative risk of death after complications identified during induction (1 in 10 cases), maintenance (0 in 8 cases) and recovery (6 in 28 cases) together with the data from the previous 5-year period.

There were no cases of *permanent morbidity* in the second 5-year period. There have been two inquests, one into a death after induced hypotension and the other into a death after aspiration during induction for a cataract extraction; both were deemed to be 'accidental deaths'.

Complications which were wholly or partially avoidable

Fourteen out of the 46 cases were considered to be in this category. This is the same number as in the previous 5

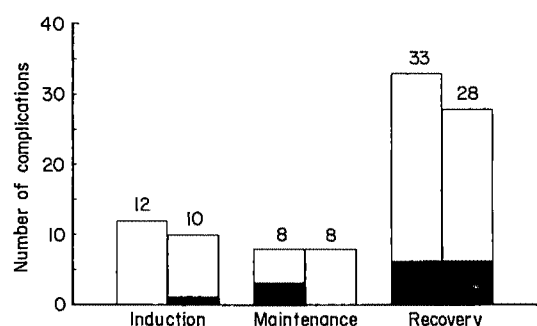


Fig. 1. Time of identification of complication in relation to anaesthetic sequence for both 5-year periods. Shaded area = deaths; one after induction in the second 5 years, three during maintenance in the first 5 years, six during recovery in both periods.

Table 2. Causes of complications identified during induction.

	Number	Deaths	Avoidable factors present
Cardiac arrest	1	0	1
Aspiration	2	1	1
Difficult intubation	1	0	0
Suxamethonium apnoea	5	0	0
Hypersensitivity	1	0	0
Totals	10	1	2

years though the proportion was better then, 14/53. However, this may reflect more rigid criteria associated with the higher expectations of the authors as a result of progress and improved standards within the specialty. Seven of the 14 cases were emergencies.

During induction (Table 2)

Cardiac arrest. A 72-year-old woman with a history of ischaemic heart disease and rheumatic fever was scheduled for anterior resection of the rectum. She had a cardiac arrest in the anaesthetic room shortly after induction. She was successfully resuscitated, the operation was abandoned and she was transferred to ICU, where she made an uneventful recovery and was discharged next day. It was evident on review of ECGs, that the patient had suffered a myocardial infarction within the previous month. The anaesthetist was a post-fellowship registrar.

Regurgitation and aspiration. A male patient of 71 years had an uneventful anaesthetic for transurethral resection of the prostate. Four days later he was anaesthetised by a consultant for a bladder washout for clot retention. He had had a haematemesis during the previous night. He regurgitated on induction and aspirated altered blood. His chest X ray and oxygenation were affected, but he recovered after 26 hours in ICU. A rapid sequence induction with intubation, perhaps preceded by gastric aspiration, would probably have averted this problem.

During maintenance (Table 3)

Cardiac ischaemia and hypotension. A 56-year-old man for elective nephrectomy, who took diuretics for heart failure, was hypotensive throughout the procedure (systolic blood pressure 70–90 mmHg) and was admitted to ICU with a provisional diagnosis of myocardial infarction. Initial treatment with both dobutamine and dopamine infusion led to his discharge after 3 days. Further elaboration of his history and investigation revealed a likely myocardial

Table 3. Causes of complications identified during maintenance.

	Number	Deaths	Avoidable factors present
Cardiac ischaemia, arrhythmia or arrest	4	0	2
Bronchospasm	3	0	0
Aspiration	1	0	0
Totals	8	0	2

Table 4. Causes of complications identified during recovery.

	Number	Deaths	Avoidable factors present
Ventilatory failure	20	3	8
Aspiration	3	0	0
Myocardial ischaemia	1	1	1
Respiratory obstruction	1	0	0
Respiratory or cardiac arrest	2	1	1
Brain death	1	1	0
Totals	28	6	10

infarction within the preceding 2 months with development of a ventricular aneurysm. The anaesthetist was a consultant.

Asystole during eye surgery. A 66-year-old diabetic patient with a history of myocardial infarction was scheduled for cataract extraction and premedicated with temazepam only. He developed a profound bradycardia that led to asystole intra-operatively. The operation continued after restoration of cardiac output using atropine, adrenaline and cardiac massage. The patient was discharged from ICU next day. Bradycardia is a common occurrence on surgical manipulation of the eye and might not have occurred if a more appropriate premedication had been used. The anaesthetist was a consultant.

During recovery (Table 4)

Eight patients failed to breathe adequately after reversal and we consider should have had elective ventilation of their lungs.

Chest infection. A patient failed to breathe adequately after an uneventful anaesthetic for elective exploration of small bowel fistula. There was documented evidence of a chest infection before operation, treatment of which might have prevented a 2-day stay in ICU. The anaesthetist was a consultant.

Central and peripheral ischaemic disease. A 63-year-old woman underwent laparotomy for bowel obstruction and 1.5 litres of free fluid was drained from the peritoneal cavity. She had a history of left ventricular failure and peripheral vascular disease. Reintubation of the trachea was needed in Recovery before transfer to ICU. An episode of supraventricular tachycardia next day resolved with DC shock but she remained hypotensive and died 2 days later. A period of elective ventilation of the lungs of a patient with an upper abdominal incision and severe cardiovascular disease might have averted this death after a benign surgical condition. The anaesthetist was a senior registrar.

Kyphoscoliosis, restrictive lung disease and polymyalgia. An 80-year-old patient with kyphoscoliosis and polymyalgia required reintubation in Recovery after a hiatus hernia repair via the abdominal route. She had a residual pneumothorax and required drainage together with overnight ventilation after which she made a steady recovery. The operation was carried out in a hospital without an ICU. The anaesthetist was a consultant.

Chronic alcoholism. An 82-year-old man with chronic alcoholism underwent a laparotomy for perforated duodenal ulcer and required tracheal reintubation in

Table 5. Grades of staff in relation to elective or emergency cases with complications.

	<i>n</i>	Elective	Emergency	Totals
Consultant	10	23	2	25
Clinical assistant	1	0	0	0
Senior registrar	1.5	2	1	3
Registrar	4	7	7	14
Senior house officer	3	3	1	4
Totals	19.5	35	11	46

*Includes contribution of half-time senior registrars (married women).

Recovery, ventilation of the lungs for 4 days and dobutamine and dopamine infusions. A period of elective ventilation might have prevented these complications. The anaesthetist was a registrar.

Hypothermia and ventilatory inadequacy. An asthmatic 66-year-old woman underwent a hysterectomy and cystectomy which lasted 4 hours. Her core temperature was 33°C on admission to Recovery where she required tracheal reintubation. Her lungs were ventilated for 4 hours and thereafter she made an uneventful recovery. Inadequate ventilation can be associated with hypothermia and could have been averted with a period of elective ventilation. The anaesthetist was a consultant.

Chronic obstructive pulmonary disease, kyphoscoliosis and age. A 79-year-old woman with chronic obstructive pulmonary disease (COPD), asthma and kyphoscoliosis failed to breathe adequately after a 3-hour laparotomy for bowel obstruction. She made a steady recovery after an initial period of ventilation but relapsed 24 hours after discharge to the ward and died. Many adverse factors in this case should have alerted the senior house officer anaesthetist to the likely early postoperative complication.

Endotoxaemia and age. Seventy-five cm of gangrenous bowel, the result of a strangulated inguinal hernia, were resected from a septic 81-year-old, 4 days after her admission to hospital. She became hypotensive and hypoxic in Recovery, was re-intubated and transferred to ICU where she died 5 days later from endotoxic shock. Elective ventilation might not have altered this course, but it should have been obvious to the anaesthetic registrar that this patient was unlikely to breathe adequately after operation.

Haemorrhage and age. The final patient was a 72-year-old alcoholic with an admission Hb of 3.9 g/dl after a haematemesis and melaena. He had already become apnoeic after sedation by a surgeon for gastroscopy, and needed tracheal intubation and controlled ventilation, an

emergency gastrectomy was then performed. He was hypotensive (after 6 units of blood and 2 litres of plasma expander) and sleepy in Recovery, his trachea was again intubated and he was transferred to ICU. The next morning he suddenly went into complete heart block and died. Planned ventilation might not have saved his life, but his need for reintubation in Recovery could have been predicted from his early course. The anaesthetist was a consultant.

The two remaining patients breathed adequately initially after operation, but required tracheal reintubation in Recovery and would have been better ventilated electively.

Peptic perforation, great age and atrial fibrillation. A woman aged 88 years had a respiratory arrest in Recovery after laparotomy for perforated duodenal ulcer. A DC shock was given for fast atrial fibrillation. Elective ventilation would have been wise. The anaesthetist was a registrar.

Chronic obstructive pulmonary disease, ischaemic heart disease and age. A lengthy emergency embolectomy was performed on an 80-year-old with ischaemic heart disease, atrial fibrillation and COPD who was breathless at rest. His need for controlled ventilation after operation was not surprising but even elective ventilation would probably not have averted his sudden death from a pulmonary embolism next morning. The anaesthetist was a registrar.

Grades of staff

The involvement of anaesthetists of different grades in relation to elective/emergency patients having complications are given in Table 5.

Fourteen cases were considered to have avoidable components. All were the consequences of actions or decisions by the anaesthetists concerned. Half of these cases were anaesthetised by consultants. Seven of the cases were after anaesthesia for emergency surgery (there were 11 emergencies in the study) (Table 6).

Complications which were unavoidable or unpredictable

During induction (Table 2)

Suxamethonium apnoea. Five patients were shown to have abnormal cholinesterase activity after apnoea after suxamethonium. They and their relatives were investigated and advised.

Aspiration. An 81-year-old man for an elective cataract extraction regurgitated bile on induction and aspirated. He developed adult respiratory distress syndrome (ARDS) and died 18 days later. His preparation before operation was routine.

Table 6. Grades of staff in relation to the time of identification and elective or emergency status of patients with complications considered to be avoidable.

Grade	<i>n</i>	Induction	Maintenance	Recovery		Total complications
				Elective	Emergency	
Consultant	10	1	2	3	1	7
Clinical assistant	1	0	0	0	0	0
Senior registrars	1.5	0	0	0	1	1
Registrars	4	1	0	0	4	5
Senior house officers	3	0	0	0	1	1
Totals	19.5	2	2	3	7	14

Difficult intubation. A registrar was unable either to ventilate the lungs by mask or to intubate the trachea of a patient with a history of ischaemic heart disease who was to have an incisional hernia repaired. A consultant was called from another theatre and successfully intubated the trachea. The ECG showed some transient ST depression. He went into ventricular fibrillation 5 minutes after uneventful extubation, but was successfully resuscitated, transferred to ICU and made an uneventful recovery with no evidence of new myocardial infarction or of neurological deficit.

Hypersensitivity reaction. A 14-year-old girl with no previous exposure to anaesthetic agents was scheduled for genioplasty. Immediately after intubation the lungs could not be inflated and she became cyanosed, pale and pulseless. She was rapidly and successfully resuscitated, the operation was abandoned and she was transferred to ICU where she made a complete recovery although she showed initial signs of cerebral irritation. This was considered after investigation to be an atypical response to quaternary ammonium compounds involving histamine release. The patient had received tubocurarine 3 mg in order to prevent suxamethonium pains.

During maintenance (Table 3)

Aspiration. Bile was seen in the airway at the end of a mask anaesthetic for manipulation of a forearm fracture in a 2-year-old boy. The patient was turned, sucked out and admitted to ICU as a precaution. There were no sequelae and he was discharged the next morning.

Bronchospasm. A 44-year-old went into severe bronchospasm which led to the abandonment of a total hip replacement. She was discharged 8 hours later after bronchodilator therapy.

Bronchospasm also occurred in a 22-year-old during an elective tympanoplasty. She quickly recovered after the administration of aminophylline and hydrocortisone.

The third patient in this category was an 8-year-old asthmatic treated with bronchodilators, who nevertheless developed severe bronchospasm during an elective open reduction of wrist fracture. He was ventilated overnight and made a good recovery after salbutamol nebulisers.

Arrhythmias. Ventricular tachycardia developed in a fit 79-year-old during transurethral resection of the prostate. The arrhythmia ceased spontaneously and he was discharged after 4 hours in ICU.

A 70-year-old woman with ischaemic heart disease on a calcium channel blocker developed bradycardia with hypotension during a cholecystectomy. She was given atropine and then required an isoprenaline infusion for 19 hours before normal sinus rhythm was re-established.

During recovery (Table 4)

Aspiration. A patient aspirated on the ward for no obvious reason the day after an emergency colectomy. She developed mild ARDS but survived this and a femoral embolectomy the day after admission; she was returned to the ward after 8 days.

A 55-year-old diabetic regurgitated and possibly aspirated in Recovery after manipulation of a fractured ankle. She was admitted overnight but discharged next day with no sequelae. She had been starved for 6 hours.

Table 7. Clinical problems of 20 patients with ventilatory failure after operation.

Clinical problems	Number of patients
Cardiovascular disease	4
Respiratory disease	5
Bowel obstruction or strangulation	4
Septicaemia	1
Carcinomatosis	2
Perforation of viscus	3
Peripheral vascular disease	1
Kyphoscoliosis	3
Rheumatoid arthritis	1
Alcoholism	2
None of the above	5
One of the above	9
Two of the above	4
Three of the above	2
More than three of the above	0

Aspiration of bile occurred in Recovery after a patient's 50th dilatation for benign oesophageal stricture despite being positioned on her side. She did not develop ARDS and was discharged next day.

Respiratory arrest. An elderly diabetic arrested in the recovery ward after elective debridement of a gangrenous foot. Circulatory inadequacy developed but resuscitation measures restored her cardiac output. However, she remained hypotensive, never regained consciousness, and died 6 days later.

Respiratory obstruction. A 7-year-old girl developed an inspiratory stridor after extubation at the end of a ureterolithotomy. Examination revealed very large tonsils and she was admitted to ICU as a precaution until the next day.

Brain death under anaesthesia. A 62-year-old man failed to wake up after an elective mastoid operation under induced hypotension. He had been fit and well beforehand and was given standard premedication and anaesthesia. His lowest recording of (direct) systolic blood pressure was 90 mmHg during the operation. There was continuous monitoring including inspired oxygen, pulse oximetry and end-tidal CO₂ together with hard copy recordings. No unusual events were recorded during the operation. However, at the end of the procedure, the patient made no spontaneous attempts at respiration and had fixed dilated pupils. Cerebral and brainstem death was confirmed both clinically

Table 8. Surgical procedures undergone by 20 patients with ventilatory failure.

Procedure	Number of patients
<i>Emergency</i>	
Laparotomy	6
Embolectomy	1
<i>Elective</i>	
Transurethral resection of prostate	1
Laparotomy	4
Fixation of ankle	1
Oesophagoscopy	1
Dental extraction	1
Anterior repair	1
Biliary stent insertion	1
Hiatus hernia repair	1
Embolectomy	1
Cystectomy and hysterectomy	1

and electrically. A subsequent inquest recorded a verdict of accidental death. The anaesthetist was a consultant.

Ventilatory failure after attempted reversal of competitive (non-depolarising) muscle relaxants. This was the largest single group of admissions (20/46). Twelve were probably unavoidable; the other eight are discussed above in the section on avoidable complications.

The average age of this group as in the first study, was 70 years (SD 14, range 22–83) while that of the remaining 26 patients was 51 years (SD 28, range 2–88). Seven of the 20 cases were emergencies and 13 were elective. Six of the seven emergencies had laparotomies (640 such operations were performed in the 5-year period). Table 7 shows the clinical problems in the whole group and the range of operations performed on them is shown in Table 8.

Two, of the 12 patients originally classified as unavoidable, subsequently had interesting developments. The first patient, who had a rigid oesophagoscopy for dysphagia and went into ventilatory failure, was diagnosed 2 months later as having myasthenia gravis. The second patient, who had an elective anterior repair and went into ventilatory failure after operation, was readmitted 3 weeks later with ascites and died of ovarian carcinomatosis, previously undiagnosed.

The maximum length of time any of these 12 patients stayed in ICU was 47 hours (mean 21, SD 13; range 3–47) whereas, the other eight stayed an average of 42 hours (SD 22; range 11–132).

Discussion

Derrington and Smith¹ reviewed studies of anaesthetic risk, morbidity and mortality. Our first publication² highlighted the difficulties of interpretation and comparison between different studies with different selection, measuring and outcome criteria for morbidity. Cooper³ has also reviewed the seminal literature up to 1988 and re-emphasises these difficulties.

This longitudinal denominator study which started in 1979 in the same Health District eliminated the effect of some of these variables. The result is a study of evolving practice in a single environment. The evolutionary process is of course affected by staff changes: during the 10 years three Consultants have left and been replaced, SHOs have been replaced on a yearly basis as has the rotating Senior Registrar, and the Registrars changed every 2 years. This means that lessons, particularly at junior level, are less effective or transmissible than if the anaesthetic staffing remained stable and senior. Furthermore, it must be recognised that since 1979 the criteria for the definition of acceptable practice have also evolved. Equally the assessment criteria for the definition of what might be considered to be an avoidable anaesthetic-related incident have probably hardened.

Over the period monitoring equipment has improved by an order of magnitude in quality and specification and the routine employment of such devices has increased. We consider that this has made a significant impact on the reduced incidence of complications in this half of the study period.

The relevance of newer relaxants or anaesthetic drugs is almost certainly unimportant since the avoidable incidents especially are related to the manner in which anaesthesia is carried out rather than the specific effects of drugs.

The demographic profile for the District shows that the average age increases over 10 years by 11 months, hardly enough to make a big impact. The ages of the admissions in the two periods were unaltered as was the distribution between the sexes, and the mean length of stay was the same for patients staying less than 75 hours. The success of Intensive Care shows, from our own experience, that surgeons will take on more risky and elderly cases in the full expectation of an eventual satisfactory physiological outcome.

Most pressure on anaesthetists has come, in fact, from an increase in the number of cases done rather than the increase in the number of elderly at risk. The skills of anaesthetists have become more diluted as a result and supervision will decrease even further if we are not careful.

It is gratifying that, on the positive side, despite the pressure on anaesthetists, and helped by planned Intensive Care, the incidence of anaesthetic complications in the second 5 years has decreased.

A significant point to emerge from the comparison is that the number of patients admitted in an unplanned manner after emergency procedures has halved. We consider that this is partly as a result of improved assessment of patients and partly better management strategies including elective ventilation. In the first 5 years the risk of unplanned admission after emergency operations was seven times that after elective procedures; in the second period the risk has reduced to threefold.

A similar number of deaths occurred in both series, with the same proportion (55%) in each thought to be due primarily to the anaesthetic complication.

The phase of anaesthesia during which complications occur continues to invite discussion. It has been said that most preventable complications arise during maintenance and that this calls for greater vigilance and concentration on the part of the anaesthetist.⁴ The fewest complications in our cases were detected during maintenance and only two of these were believed to be avoidable. Our findings are surely predictable since induction and recovery are the least stable stages of the anaesthetic process, and recovery is the more prolonged of the two. The contribution of Recovery wards to the safety of patients is amply borne out in this study.

The largest single group of patients was admitted as a result of postoperative ventilatory inadequacy. Their mean age was the same as that in the first study and they had a similar spread of concurrent medical problems. The six emergency laparotomies were half the number in the first study. We suggested previously² that elective ventilation of the lungs after emergency laparotomies in patients over 70 years of age should be considered more routinely and our findings in the second 5 years indicate a trend in the right direction.

One of the causes for current anxiety is the experience or otherwise of anaesthetists involved in emergency work.⁵ It is a cause for concern to discover that an SHO had anaesthetised a patient of ASA grade 4 for an emergency laparotomy. If advice is sought from a senior then this fact must be recorded as well as acted upon.

The grades of those staff involved in partially or completely avoidable anaesthetic complications are interesting. Fourteen of the 46 complications were in this group. Half of these patients were anaesthetised by consultants, as were just over half of those with unavoidable complications.

Consultants are half of our staff but nevertheless one might hope for a lower incidence of avoidable complications in their practice because their experience is so much greater than that of the junior anaesthetists. We are aware conversely, in our DGH context, that they are more involved with high risk patients and doubt whether either complacency or over-confidence are significant factors. We must not allow the increased pressures of today's medical practice to result in a reduction of supervision of junior anaesthetists or toleration of seniors doing difficult cases without appropriate assistance.

Four of the patients admitted to ICU with avoidable complications died. However, they all had many medical problems and it is unlikely that any of the deaths could have been averted. The other three deaths occurred in patients who suffered unavoidable complications and we are reasonably confident that no patient died from lack of care by an anaesthetist.

It is interesting that in the 10 years three of the 16 deaths were related to induced hypotension. The two deaths in the first 5-year period were considered to be avoidable; one patient had arteriosclerosis and the other an operation for which induced hypotension is probably unnecessary. The patient in the current study, as already discussed, failed to awake without discernible warning. Many studies have been done on induced hypotension, but no actual proof exists that it improves the patients' prognosis from surgery, while complications abound. Many surgeons request it on the grounds that it improves conditions of operation and reduces blood loss. Perhaps the time has come to consider its abandonment altogether or, at the very least, to use it solely in fit patients where it is of proven value.

There is an apparent increase in complications in the last 2 years of the study (26 of the 46); we shall be continuing to monitor the figures to determine whether this is a real trend or a result of the operation of random factors. Eleven of the 14 avoidable complications also occurred in this time which is further disquieting.

The vast majority of anaesthetics are given safely and successfully, but this study has raised considerations which may help to improve anaesthetic practice. This is likely to become even more important as we are presented with even older and sicker patients to anaesthetise since we know from this study that the average age of those who died was 73 while that of the remainder was 57 years of age.

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Behaviour of small children before induction

The effect of parental presence and EMLA and premedication with triclofos or a placebo

B. PAGE AND J. O. MORGAN-HUGHES

Summary

The effect of the prescription of triclofos 70 mg/kg orally 90 minutes before operation on the behaviour of 263 children aged 1 to 5 years was studied in a blind, randomised, placebo-controlled trial. EMLA was applied to the dorsum of one hand when the premedication was administered, and all the children were accompanied by a parent during induction of anaesthesia. The frequency of satisfactory behaviour was significantly higher in the triclofos group ($p < 0.02$), but the time spent in the recovery ward was increased.

Key words

*Premedication; triclofos, parental presence.
Anaesthetics, local; eutectic mixtures.*

The frequency of calm behaviour at induction of anaesthesia of unsedated younger children is lower than that of children over the age of 5 years,^{1,2} and arterial oxygen desaturation is more likely to occur during induction of anaesthesia in younger children, particularly if the child is agitated.³ In addition, there is evidence that parental presence at induction is helpful to children aged 2 to 6 years⁴ and that the use of a eutectic mixture of local anaesthetics (EMLA) reduces the pain of venepuncture in children aged 1 to 5 years.⁵ The literature lacks studies designed to show whether sedative premedication improves the behaviour of small children who have the benefit of EMLA and a parent present at induction.

The oral route is preferred, in many countries, for the painless administration of sedative premedication for most scheduled surgery.⁶ Which drug is most likely to succeed is not clear. Trimeprazine, which is favoured by some British anaesthetists,⁶ was shown to increase the frequency of crying and struggling at induction when compared to a placebo.^{7,8} Benzodiazepines are widely used for sedation before operation in children⁶ but triclofos was found to produce more satisfactory behaviour at induction than diazepam^{9,10} or flunitrazepam.¹⁰ Another less palatable form of chloral hydrate was a better sedative than midazolam in a study of children under 5 years.¹¹

The oral administration of 0.7 ml/kg of triclofos 100 mg/ml with atropine 90 minutes before anaesthesia

increases the pH and has no significant effect upon the volume of gastric contents when compared to intramuscular pethidine and atropine.¹⁰

A literature search has revealed no placebo-controlled trials of the use of triclofos in this way.

The object of this study is to determine whether triclofos 70 mg/kg alters the frequency of calm behaviour of young children before induction who have a parent present and the benefits of EMLA.

Methods

Children aged 1 to 5 years were studied. The protocol was approved by the hospital ethics committee and informed written consent was obtained from parents.

The children were scheduled for general, genito-urinary, plastic, orthopaedic, ophthalmic or dental surgery under general anaesthesia as day cases or inpatients. Day cases were defined, for the purposes of the study, as children anaesthetised on the day of admission. The type of surgery was defined by the operation and not by the specialty of the surgeon; for example a circumcision was defined as a genito-urinary operation even when it was performed by a general surgeon. Children who were ASA grade 1 or 2, judged fit for anaesthesia, and whose parents were able and willing to accompany them to the anaesthetic room were

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Accepted 27 February 1990.

considered for inclusion in the study. Children who were educationally subnormal or receiving sedative drugs were excluded, as were those who had a second anaesthetic on the same admission to hospital. The child was excluded if an investigator was unable to see the child and parent beforehand with adequate time for premedication.

The investigator recorded the date of any previous admission during which a general anaesthetic was administered and made a prediction as to whether the child would cry in the anaesthetic room.

The bottles that contained the premedication, the contents of which were assigned using a computer-generated random number program, were prepared by the pharmacy and identified by number only. They contained 20 ml of either atropine (Macarthy's Medical Ltd., Romford) 0.05 mg/ml or triclofos (Evan's Medical Ltd., Bedfordshire) 100 mg/ml and atropine 0.05 mg/ml. The solutions were made similar in appearance and taste by the addition of vanilla, orange and yellow colouring and the bottles' contents were known only to the pharmacist until the trial was completed. A dose of 0.7 ml/kg to a maximum of 15 ml was prescribed to be administered orally 1.5 hours before operation. The children were admitted sequentially to the trial so that the first child to meet the inclusion criteria was allocated bottle number one and so on.

The nurse who gave the premedication applied EMLA on the dorsum of a hand at the same time, noted the time and recorded whether the premedication was easy, difficult or impossible to administer.

The ward data record sheet was retained on the ward and a separate sheet accompanied the child to theatre.

The child's demeanour in the anaesthetic room and response to induction were assessed by the anaesthetist using a method modified from that of Doughty¹ to allow assessment of inhalation inductions.

The terms used to describe the child's demeanour on arrival in the anaesthetic room were: sleepy, cheerful, serious, apprehensive, tearful or noisy. The options for describing the response to intravenous induction were: no response, winced, whimpered, cried or violent, and to inhalation induction were: smooth, minor objection or needed restraint. The anaesthetist was asked to tick one term to describe the child's demeanour and one to describe the response to induction.

The investigators classified a sleepy, cheerful or serious demeanour as satisfactory for statistical purposes. No response, or winced, were deemed satisfactory responses to intravenous induction. Smooth or minor objection described a satisfactory response to inhalation induction. If more than one induction method was attempted only the response to the first method was analysed. Satisfactory behaviour was defined by both a satisfactory demeanour and a satisfactory response to induction.

No attempt was made by the investigators to influence the anaesthetic management.

The time of admission to, and discharge from, the recovery ward and whether the child vomited in the recovery ward was recorded by the recovery nurse.

The size of the trial was guided by a statistical method described by Pocock¹² and by other studies using Doughty's method of assessment. The terms used to describe behaviour were looked upon as independent variables, and Chi-squared tests were used for analysis. Premedication and recovery times were analysed by unpaired *t*-test.

Results

Three hundred and fifty-eight children in the appropriate age group admitted consecutively for surgery were considered for inclusion in the trial during a 7-month period from January 1989. Two hundred and seventy of these children met the inclusion criteria, but seven were withdrawn from the trial before analysis, four because a parent did not accompany the child to the anaesthetic room. One child was incorrectly weighed and, as a consequence, the wrong dose of premedication was administered. The operation planned for one child was cancelled, after the premedication was given, to allow an emergency admission access to theatre. Failure to record behaviour caused a seventh child to be withdrawn. Of the 263 children remaining, 128 were in the triclofos group and 135 were placebo controls. Two hundred and eighteen children were accompanied by their mothers and 45 by their fathers.

Age

Table 1 shows the age distribution of the children studied and the variation in behaviour with age.

Previous general anaesthesia

One hundred and ninety-two children had no anaesthetic history and 126 had a general anaesthetic on a previous admission to hospital. Sixty-six percent of the former behaved satisfactorily compared to 51% of the latter.

Prediction of behaviour

An investigator predicted that 82 children would cry and that 178 would not. No prediction was made in three cases. Forty-three children behaved satisfactorily in the group in which crying was predicted and 118 behaved satisfactorily in the group in which no crying was predicted.

Daycases and inpatients

There were 115 daycases and 148 inpatients. Eighty-four (73%) of the day-case children behaved satisfactorily compared to 78 (53%) of inpatients.

The anaesthetist

One hundred and eighteen anaesthetics were administered by two consultant anaesthetists, A and B. Other anaesthetists administered less than 20 each. Table 2 shows the frequency with which satisfactory behaviour was recorded by different anaesthetists.

Table 1. The age of the children studied and the variation of behaviour with age.

Age (years)	Number	Satisfactory number (%)
1	29	15 (52)
2	63	33 (52)
3	70	43 (61)
4	48	33 (69)
5	53	38 (72)

Table 2. The frequency of satisfactory behaviour recorded by different anaesthetists.

Anaesthetist	Number	Satisfactory number (%)
A	81	56 (69)
B	37	25 (68)
Others	145	81 (56)

Comparability of triclofos and placebo groups

There were no significant differences between the triclofos and control groups for age, incidence of previous anaesthetics, prediction of behaviour, proportion of day cases, anaesthetists or surgical specialty (Table 3).

Ease of administration

There was no significant difference in the ease with which the nurses were able to administer the two preparations (Table 4). Administration proved impossible on four occasions, two in each treatment group. These four patients were not withdrawn from the study.

Timing of administration

The mean time between the administration of the premedication and induction of anaesthesia was 105 (SD 28) minutes for the placebo group and 108 (SD 41) minutes for the triclofos group. The difference is not significant.

Demeanour

The frequency of unsatisfactory demeanour in the triclofos group was half that in the placebo group (Table 5). This difference is significant, Chi-squared = 10.1, $p < 0.005$. More children were sleepy in the triclofos group, but this did not appear to influence the anaesthetists' choice of method of induction since equal numbers of intravenous inductions were performed in each group.

Table 3. Comparison of triclofos and placebo treatment groups.

	Triclofos (<i>n</i> = 128)	Placebo (<i>n</i> = 135)
<i>Age (months)</i>		
1st quartile	32	28
Median	44	40
3rd quartile	55	56
Previous anaesthetic	37	34
Crying predicted	34	48
Day cases	55	60
<i>Anaesthetist</i>		
A or B	57	61
Others	71	74
<i>Surgical specialty</i>		
Genito-urinary	42	47
General	36	34
Plastic	26	24
Ophthalmic	21	20
Orthopaedic	3	9
Dental	0	1

Table 4. Ease of administration.

	Triclofos	Placebo
Easy	98	114
Difficult	27	17
Failed	2	2
No record	1	2

Response to induction

The frequency of unsatisfactory response to induction was lower in the triclofos group both for intravenous (Table 6) and inhalation induction (Table 7) and when the response to both forms of induction are considered together (Table 8), the difference is significant, Chi-squared = 3.97, $p < 0.05$.

Behaviour overall

Seventy percent of the children in the triclofos group had both a satisfactory demeanour on arrival in the anaesthetic room and a satisfactory response to induction (Table 9). This is significantly higher than the 54% frequency of overall satisfactory behaviour in the placebo group, Chi-squared = 6.64, $p < 0.02$.

Time in recovery ward

The mean time spent in the recovery ward by patients in the placebo group was 24 (SD 12) and 35 (SD 18) minutes in the triclofos group. This difference is significant, $p \approx 0$.

Vomiting in recovery ward

Four children in each group vomited in the recovery ward.

Discussion

Triclofos is one of a group of substances, like chloral hydrate, that depend upon trichloroethanol for their pharmacological activity. Triclofos is trichloroethyl phosphate, a stable ester of trichloroethanol, to which it is rapidly hydrolysed after administration. The use of this group of drugs as premedication for children has been reported by several authors.^{9,10,13-16} The only placebo-controlled trial was that of chloral hydrate 50 mg/kg reported by

Table 5. The number of patients with satisfactory demeanour.

Demeanour	Triclofos	Placebo
<i>Satisfactory</i>		
Sleepy	33	15
Cheerful	32	29
Serious	42	46
Total	107	90
<i>Unsatisfactory</i>		
Apprehensive	11	22
Tearful	4	14
Noisy	6	9
Total	21	45*

*Chi-squared = 10.1; $p < 0.005$.

Table 6. The number of patients who responded satisfactorily to intravenous induction.

Response	Triclofos	Placebo
<i>Satisfactory</i>		
No response	59	53
Winced	14	10
Both	73	63
<i>Unsatisfactory</i>		
Whimpered	11	15
Cried	13	15
Violent	2	3
Total	26	33

Rollason¹³ who showed no significant difference in behaviour. The dose of triclofos was approximately 70 mg/kg administered more than one hour before operation in studies in which triclofos was compared favourably to benzodiazepines.^{9,10} Gupta, Blades and Hatch¹⁵ reported the effectiveness of hyoscine and atropine as anticholinergic drugs when used as oral mixtures with triclofos 75 mg/kg.

Contrary to a report by Saarnivaara *et al.*¹¹ that triclofos will no longer be manufactured, we were assured by Evan's Medical Ltd. before this study that triclofos will be produced for as long as it is required.

The use of orange colouring in the preparation of the premedicants was not ideal since the mother of a 'hyperactive' child maintained that orange colourants had an adverse effect on his behaviour and she would not allow him to be enrolled in the trial. However, the base for triclofos mixture contains sunset yellow (E110), not tartrazine.

Ease of administration is an important consideration in the choice of oral premedication for children, and administration of triclofos has been cited as a problem;⁹ however, it proved no more difficult to administer than the placebo. This may merely reflect the skill with which the pharmacy matched the appearance and taste of the two treatments. The four children to whom the nurses failed to administer the premedication were not withdrawn from the trial since the object was to observe the effects of the prescription.

Another concern is that premedication may prolong the recovery time; the time spent in the recovery ward by the triclofos group was significantly longer. However, this is difficult to interpret since we did not control the type of anaesthetic given. A prolonged recovery time has implications for recovery ward staffing levels.

The assessment of the children's behaviour has a large subjective element and it would be desirable to have a single observer for all the children. The differences between

Table 7. The number of patients who responded satisfactorily to inhalation induction.

Response	Triclofos	Placebo
<i>Satisfactory</i>		
Smooth	20	19
Minor objection	4	5
Both	24	24
<i>Unsatisfactory</i>		
Needed restraint	5	15

Table 8. Number of patients who responded satisfactorily to induction.

Response	Type of induction	Triclofos	Placebo
<i>Satisfactory</i>			
	Intravenous	73	63
	Inhalation	24	24
	Both	97	87
<i>Unsatisfactory</i>			
	Intravenous	26	33
	Inhalation	5	15
	Both	31	48*

*Chi-squared = 3.97; $p < 0.05$.

anaesthetists in the frequency with which they recorded unsatisfactory behaviour may be accounted for either by differences in the behaviour of the children they observed, or by differences between the anaesthetists as observers. This limitation in the design was imposed by our resources. A generous estimate of trial size increased the chance that randomisation would reduce bias from this and other sources.

A relationship between behaviour before operation and age within the under-6 age group and the adverse effect of previous exposure to general anaesthesia on pre-operative behaviour was shown by Rosen and colleagues.¹⁷ Our observations are in accord.

The surgeons decided which children were admitted on the day of operation and the authors do not know on what basis the decisions were made. No attempt has therefore been made to analyse the extent to which other factors, such as the age of the patients, are responsible for the much higher incidence of calm behaviour in day cases. Psychological changes in children having minor surgery were investigated by Scaife and Campbell¹⁸ who randomly allocated children to day case or inpatient management. They did not examine the influence of the timing of admission on behaviour before induction and this merits further study.

We showed little ability to predict a child's behaviour. The predictions were based largely on the behaviour of the child on the ward at the interview beforehand. However, the less experienced observer predicted crying accurately more frequently than the more experienced. Hannallah *et al.* in contrast presented evidence of the greater ability of experienced staff anaesthetists to predict the pre-operative behaviour of children when compared to residents.¹⁹

Parental presence at induction has become our usual practice, particularly since the advent of EMLA, so we usually avoid sedative premedication because it is unlikely to be helpful. The finding that the frequency of satisfactory behaviour was higher in the triclofos group was therefore contrary to our expectations.

Table 9. Behaviour overall.

Behaviour	Triclofos	Placebo
Satisfactory	89	73
Unsatisfactory	39	62
Total	128	135

*Chi-squared = 6.64; $p < 0.02$.

Conclusion

The prescription of triclofos 70 mg/kg 90 minutes before operation increased the frequency of satisfactory behaviour in the anaesthetic room of children aged 1 to 5 years who were accompanied by a parent and who had the benefit of EMLA.

The mean time spent in the recovery ward was 11 minutes longer in the group who received triclofos. There was no difference in the frequency of vomiting in the recovery ward.

Acknowledgments

The authors thank Mr M. Callam, Pharmacist Production Manager, Norfolk and Norwich Hospital who was responsible for the preparation of the premedication and randomisation, Dr A. Bangham, Lecturer, University of East Anglia for statistical advice, and their medical and nursing colleagues for their cooperation.

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Intravenous fluid load and recovery

A double-blind comparison in gynaecological patients who had day-case laparoscopy

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Summary

The effect of intra-operative fluid and dextrose administration upon recovery was tested in a randomised, double-blind trial. Three groups of 25 patients, each undergoing laparoscopic examination as day cases, were studied. The two groups who received fluid (20 ml/kg compound sodium lactate solution) showed significant improvement ($p < 0.05$) in the variables that reflected hydration. The fluid group who also received dextrose (1 g/kg) exhibited further significant improvement. Intra-operative fluid and dextrose administration appears to confer some benefit upon recovery in patients who have minor surgery.

Key words

*Complications; postoperative.
Fluid balance*

Most anaesthetists suspect that the well-being and recovery of patients after anaesthesia and surgery is prejudiced by dehydration and possible starvation, and recent studies^{1,2} have suggested that patients benefit significantly from the administration of peri-operative fluid. However, some questions remained unanswered, since the relative contribution of a fluid load compared with a fluid load with calories was not studied, nor was the fluid load related to body weight, and only subjective measurements of recovery were used.

We investigated the relative effects of fluid alone compared with calorie-containing fluid, in a controlled, double-blind study with objective tests of psychomotor function during recovery.

Method

The protocol was approved by the local ethics committee. An information sheet was sent to each patient before admission, and informed, witnessed, verbal consent was obtained. Seventy-five female patients aged 18 to 40 years were studied in a double-blind, randomised, single-centre trial. They were ASA 1 or 2, took no routine medication and underwent day-case laparoscopic examination.

Day case laparoscopic examination was chosen to provide a population with a relatively high rate of side effects³ and one might expect any major influence or

improvement to be significant for the numbers studied. Patients were seen before operation by one of the authors for baseline assessments and to enable demographic details to be recorded. No premedication was prescribed. Each patient was allocated at random to one of three groups.

The patients were usually in the morning theatre session, and at worst would endure a period of approximately 16 hours' fast, from 2100 hours the previous night until 1300 hours in the afternoon after surgery. The control group received no peri-operative fluid, the compound sodium lactate (CSL) group 20 ml/kg compound sodium lactate solution, and the compound sodium lactate solution/dextrose (CSL/dext) group 20 ml/kg compound sodium lactate solution with 1 g/kg added dextrose. The fluid load was based on a daily water requirement of approximately 30 ml/kg. The caloric supplement was chosen to represent a typical oral intake which would not induce hyperglycaemia.

An 18-G intravenous cannula was inserted under local anaesthesia, and connected to a premeasured bag of the designated infusion fluid. The intravenous fluid containers were covered by a large paper bag for concealment, and the infusion was given over 45 minutes. The blood glucose level was measured at the time of intravenous cannulation using a dextrostix (BM-test-glycemi, Boehringer Mannheim, W. Germany) and reflomat system (Reflomat, Boehringer Mannheim, W. Germany), which correlated well with local laboratory assays.

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Accepted 22 February 1990.

The same anaesthetic sequence of propofol (2.5 mg/kg), fentanyl (1.5 µg/kg) and vecuronium (0.07 mg/kg) was given to each patient, who was then hand ventilated with nitrous oxide and oxygen (50:50). Enflurane 1% was added and ventilation continued until relaxation was sufficient to permit intubation. Anaesthesia was maintained, after tracheal intubation, using nitrous oxide and oxygen (2:1) with enflurane 1%. A fresh gas flow of 100 ml/kg, a tidal volume of 10 ml/kg using a Bain system and an anaesthetic ventilator (Penlon, Nuffield series 200) were used throughout. The enflurane was stopped, 100% oxygen was administered and neostigmine 2.5 mg with glycopyrronium 0.5 mg given to reverse neuromuscular block, after the last stitch had been inserted.

Surgery comprised laparoscopic examination of pelvic viscera without any additional procedures. Any departure from this was cause for exclusion from the study. A need for fluids beyond that stipulated in the protocol and a contraindication to any of the proposed anaesthetic drugs also caused exclusion.

Codeine 45–60 mg intramuscularly, paracetamol 1 g orally and metoclopramide 10 mg intramuscularly were prescribed as required in the postoperative period. The data for continuous values were analysed using a two-tailed Student's *t*-test; for noncontinuous the Chi-squared and Fisher's exact test were used and the probability was $p < 0.05$.

A Trieger test⁴ was performed before operation by one of the authors and a questionnaire was completed on the symptoms of nausea, vomiting, dizziness, hunger, thirst, drowsiness, headache, sore throat, abdominal pain and faintness on standing.

Eye opening on command was tested at one-minute intervals: the ability to give date of birth correctly and the times from the end of surgery were recorded. A Trieger test was performed at 15, 60, 120 and 180 minutes after operation. The presence of the above symptoms was recorded once again at 3 hours after operation as well as the patient's assessment of the experience and her readiness to go home. The observer also assessed readiness for discharge using the criteria of a steady gait and general wellbeing. Any medication received after operation was recorded together with urine output and food or fluid intake. Both observer and patient graded readiness for discharge as: ready immediately; delayed until fit; or to be detained overnight.

The patient at discharge was given a questionnaire on which she was asked to record any of the above symptoms on each of the first 3 postoperative days, and when she

Table 1. Demographic data; mean (SD).

	Control <i>n</i> = 24	CSL <i>n</i> = 24	CSL/dext <i>n</i> = 25	Total <i>n</i> = 73
Age; years	31.5 (6.2)	31.4 (6.5)	32.9 (6.5)	32.0 (6.31)
Weight; kg	62.4 (10.9)	60.6 (8.4)	62.2 (11.9)	61.7 (10.4)
Duration of operation; minutes	19.8 (4.7)	21.8 (6.6)	18.9 (7.0)	20.1 (6.2)
Pre-operative glucose; mmol/l	4.5 (1.7)	4.6 (1.2)	4.4 (1.3)	4.5 (1.4)

There was no statistical difference between groups.

resumed her normal daily activities. Assessments were made by one of the authors who was blinded to the treatment groups and did not participate in administration of the anaesthetic.

Results

Table 1 lists the demographic data. Duration of operation was defined as the time from induction of anaesthesia to the time of the final suture, when 100% oxygen and the reversal agents were given. The means and standard deviations for each variable and group are listed; there was no significant difference between the groups. Comparison of the groups with respect to the incidence of pre-operative symptoms (Table 6) reveals no significant difference, except in the incidence of headache in the CSL group when compared to the CSL/dext but not the control group.

The initial measures of recovery were the periods from the end of surgery to eye opening in response to command, and ability to give date of birth correctly. There was no significant difference between groups for either of these; the overall mean time to eye opening was 4.3 (SD 2.3) minutes and the overall mean time to giving date of birth was 8.7 (SD 2.6) minutes.

We scored the number of dots missed with the Trieger test on each occasion (Table 2). There was no significant difference between any of these groups or times, except that the 15-minute values were significantly different ($p < 0.001$) from scores at all other times. Table 3 lists the blood glucose levels of each group before and after operation; there was no statistically significant difference between the groups or between the pre- and postoperative levels within each group.

The incidence of the use of analgesics and antiemetics is listed in Table 4. There was no significant difference

Table 2. Trieger test; number of dots missed mean (SD).

	Control <i>n</i> = 24	CSL <i>n</i> = 24	CSL/dext <i>n</i> = 25	Total <i>n</i> = 73
<i>Trieger test</i>				
Before operation	2.5 (3.9)	2.3 (3.2)	1.8 (5.4)	2.2 (4.2)
15 minutes after operation	14.8 (10.7)	15.5 (10.0)	14.7 (9.6)	15.0 (9.9)
60 minutes after operation	3.8 (4.6)	4.3 (4.8)	4.4 (5.9)	4.2 (5.1)
120 minutes after operation	2.5 (3.5)	2.3 (3.3)	2.6 (4.3)	2.5 (3.7)
180 minutes after operation	2.1 (3.9)	1.9 (2.8)	2.2 (3.9)	2.1 (3.6)

*The differences in 15-minute values was highly significant compared to pre-operative values ($p < 0.001$).

Table 3. Glucose levels before and 3 hours after operation, mean (SD).

	Control <i>n</i> = 24	CSL <i>n</i> = 24	CSL/dext <i>n</i> = 25	Total <i>n</i> = 73
Glucose before operation; mmol/l	4.45 (1.74)	4.58 (1.23)	4.42 (1.34)	4.5 (1.4)
Glucose after operation; mmol/l	4.80 (1.36)	4.40 (1.29)	4.90 (2.10)	4.7 (1.6)

There was no significance between groups or between pre- and post-operative levels.

between the groups in the use of these drugs, although the CSL group had a significantly ($p < 0.05$) greater proportion of patients who required no medication compared with control. We noted, as a crude indicator of hydration and thirst, the numbers who drank in the first 3 hours after operation. Significantly fewer patients in the CSL group (19 of 24) drank than in the control (24 of 24) or the CSL/dext groups (23 of 25), $p < 0.05$.

Fitness for discharge was assessed at 3 hours after operation by the blinded observer and the patients themselves (Table 5). The CSL/dext group had significantly more patients ready for immediate discharge, as assessed by the observer but not by the patient.

Three hours after operation the patient's overall assessment of side effects did not differ significantly between the groups. Table 6 lists the percentages and numbers in each group who reported the various symptoms before operation, 3 hours after operation, and on days 1, 2 and 3. The only significant pre-operative difference lay in the incidence of headache between the CSL and CSL/dext groups. Three hours after operation the groups were only distinguishable in that comparison of the CSL group with the control group gave a significantly lower incidence of faintness on standing.

On day 1 after operation the CSL/dext group had a significantly lower incidence of dizziness, drowsiness and faintness on standing when compared with the control but not the CSL group. No other statistical difference was seen. On day 2 the CSL/dext group had a significantly lower incidence of headache and faintness on standing in comparison with control. This group had a significantly lower incidence of sore throat compared with the CSL but not the control group. There were no other statistically significant variations between the groups with respect to other symptoms on either day 2 or 3.

Table 7 lists the numbers in each group according to the day they resumed their normal activities (or were still to do so), i.e. day 1, 2 or 3. No difference between the groups was significant.

Table 4. Administration of medication after operation; number in group (%).

	Control <i>n</i> = 24	CSL <i>n</i> = 24	CSL/dext <i>n</i> = 25	Total <i>n</i> = 73
Antiemetic	3 (13)	1 (4)	5 (28)	9 (13)
Codeine	16 (67)	12 (50)	14 (61)	42 (59)
Paracetamol	8 (33)	3 (13)	4 (17)	15 (21)
No medication	1 (4)	9 (37)*	6 (26)	16 (23)

*Significant compared with control group ($p < 0.05$).

Table 5. Assessment for discharge at 3 hours after operation by observer and patient; number in group (%).

	Control <i>n</i> = 24	CSL <i>n</i> = 24	CSL/dext <i>n</i> = 25	Total <i>n</i> = 73
<i>Observer's assessment</i>				
Discharge at 3 hours	10 (42)	14 (58)	18 (75)*	42 (59)
Delayed discharge	14 (58)	9 (38)	6 (25)*	29 (40)
Stay overnight	0 (0)	1 (4)	0 (0)	1 (1)
	<i>n</i> = 24	<i>n</i> = 24	<i>n</i> = 25	<i>n</i> = 73
<i>Patient's assessment</i>				
Discharge at 3 hours	5 (21)	7 (29)	10 (40)	22 (30)
Delayed discharge	12 (50)	15 (63)	12 (48)	39 (53)
Stay overnight	7 (29)	2 (8)	3 (12)	12 (17)

*Significant compared with control group $p < 0.05$.

Discussion

The question of intra-operative fluid administration to patients who have minor surgery remains controversial. Most of the previous work about fluid therapy and surgery relates to patients undergoing major surgery. Shires *et al.* in 1961¹ concluded that the extracellular fluid compartment was contracted as a result of 'third space' losses proportional to the degree of surgical trauma. This work supported the requirement for fluid beyond that indicated by readily measurable or calculable losses during major surgery. Subsequently, authors have supported this view, and cited benefits such as enhanced peri-operative haemodynamic stability,⁶ prevention of renal failure, moistening of bronchial secretions⁷ and less thirst after operation.⁸ More recently, however, Twigley and Hillman⁹ questioned this approach, in its extrapolation to minor surgery, in the light of the increased secretion of antidiuretic hormone inherent in the stress response to surgery. This view is supported by Sinnatamby *et al.*¹⁰ who documented that the degree of antidiuretic hormone secretion was unaffected by increased fluid administration in patients who have major surgery.

In 1986 Keane and Murray,¹ in a nonrandomised, unblinded trial showed that peri-operative fluid administration to patients undergoing minor surgery enhanced recovery with, in particular, a dramatic reduction in the incidence of postoperative drowsiness and thirst. Spencer² in 1988 also showed some minor benefit from this approach in patients who have minor gynaecological surgery. However, neither of these authors considered the question of caloric compared with fluid supplementation, nor related the fluid administered to body weight.

Our results lend support to fluid administration in minor surgery. Consideration of symptoms (Table 6) showed that significant increase occurred within the groups compared before operation and then persisted as long or longer in the control untreated group. Specifically dizziness, nausea and faintness on standing all persisted longer in the control group. Those who received fluid showed significant improvement in the variables that reflect hydration, although the incidence of thirst *per se* did not differ between the groups. They also tended to require less post-operative medication, and at assessment for discharge appeared to be more ready to go home than the control group. In addition, the group that also received dextrose showed improvement in the incidence of postoperative dizziness, drowsiness and sore throat at the times indicated

Table 6. Incidence of symptoms. Control (C) $n=24$; CSL $n=24/23$; CSL/dext $n=25$. Number in each group (%).

Symptom		Before operation	3 hours after operation	Day 1	Day 2	Day 3
Nausea	C	0 (0)	13 (54)*	5 (21)*	0 (0)	0 (0)
	CSL	2 (8)	11 (46)*	5 (22)	1 (4)	0 (0)
	CSL/dext	3 (12)	12 (48)*	1 (5)	0 (0)	0 (0)
Vomiting	C	0 (0)	4 (17)	2 (8)	0 (0)	0 (0)
	CSL	1 (4)	4 (17)	1 (4)	0 (0)	0 (0)
	CSL/dext	0 (0)	6 (24)*	1 (5)	0 (0)	0 (0)
Dizziness	C	0 (0)	11 (45)*	8 (33)*	5 (21)*	3 (13)
	CSL	1 (4)	6 (25)*	4 (17)	1 (4)	1 (4)
	CSL/dext	0 (0)	8 (32)*	1† (5)	2 (9)	0 (0)
Hunger	C	15 (63)	8 (33)*	5 (21)*	2 (8)*	1 (4)*
	CSL	13 (54)	10 (42)	3 (13)*	0 (0)*	1 (4)*
	CSL/dext	11 (44)	5 (20)	1 (5)*	3 (14)*	2 (9)*
Thirst	C	16 (67)	23 (96)*	13 (54)	4 (17)*	2 (8)*
	CSL	16 (67)	20 (83)	12 (52)	5 (22)*	2 (9)*
	CSL/dext	16 (64)	20 (80)	8 (86)	5 (23)*	2 (9)*
Drowsiness	C	0 (0)	21 (88)*	13 (54)*	4 (17)	3 (13)
	CSL	3 (13)	17 (70)*	10 (43)*	3 (13)	2 (9)
	CSL/dext	2 (8)	16 (64)*	5† (23)	3 (14)	1 (5)
Headache	C	2 (8)	2 (8)	6 (25)	7 (29)	6 (25)
	CSL	6† (25)	5 (21)	5 (22)	3 (13)	1 (4)
	CSL/dext	0 (0)	2 (8)	3 (14)	1† (5)	1 (5)
Sore throat	C	2 (8)	18 (75)*	18 (75)*	13 (54)*	9 (38)*
	CSL	0 (6)	19 (79)*	17 (74)*	14 (61)*	7 (30)*
	CSL/dext	0 (0)	20 (80)*	13 (59)*	6† (27)*	4 (18)*
Abdominal pain	C	3 (13)	20 (88)*	14 (58)*	12 (50)*	9 (38)*
	CSL	2 (8)	19 (84)*	9 (39)*	8 (35)*	6 (26)
	CSL/dext	1 (4)	20 (80)*	13 (59)*	11 (50)*	6 (27)
Faintness on standing	C	0 (0)	9 (38)*	8 (33)*	5 (21)*	3 (13)
	CSL	0 (0)	3† (13)	3 (13)	2 (9)	1 (4)
	CSL/dext	0 (0)	6 (24)*	2† (9)	0† (0)	0 (0)

*Significantly different on comparison with pre-operative level within group $p<0.05$.†Significantly different on comparison with control $p<0.05$.

(Table 6). The mechanism of this is not clear, but glucose administration has been shown to alter the hormonal profile of the stress response,¹¹ in particular leading to increased insulin secretion. The glucose levels in our groups did not differ but this may have been because the sampling interval of 3 hours missed the peak.

We used a combination of objective and subjective tests of recovery in our study. The objective results were disappointing, with no difference between the groups. It is possible that learning diminished the value of the Trieger test in the later postoperative period, for even though the

scores had returned to normal by one hour after operation it was obvious that the patients were not fit for discharge at this time.

Similarly, the discrepancy between the patient's own assessment for discharge and that of the observer (Table 5) highlights the difference between readily assessable features, such as steadiness of gait, and subjective well being. The subjective assessments were necessarily straightforward and self-explanatory because we required the patients to return a questionnaire. The response rate was 94% and it revealed the high incidence of sore throat, presumably related to tracheal intubation in all patients; the high incidence of abdominal pain undoubtedly related to the surgery and the fact that no group had completely resumed normal activities within 3 days after this supposedly trivial surgery.

Sinnatamby *et al.*¹⁰ concluded that factors other than extracellular fluid replacement influence the degree of anti-diuretic hormone release. Indeed, factors such as anxiety, motivation and surgical trauma are difficult to assess and control even in the context of minor surgery. It is possible that in spite of our assessment we did not account for perhaps the most influential factors that govern recovery and overall wellbeing, and the limited numbers may have

Table 7. Resumption of normal activities; number in group (%).

	Control $n=24$	CSL $n=23$	CSL/dext $n=21$	Total $n=68$
Resumption of normal activities				
Day 1	1 (4)	2 (9)	3 (14)	6 (10)
Day 2	11 (46)	6 (26)	7 (33)	24 (35)
Day 3	9 (37)	12 (52)	9 (43)	30 (44)
Not yet	3 (13)	3 (13)	2 (10)	8 (12)

There was no statistical difference.

failed to reveal other more subtle differences between the groups.

Conclusion

We have demonstrated a number of differences between the groups because of the number of comparisons made, although a p value of < 0.05 is too high to conclude unequivocally that a real difference exists. However, we believe that a trend is identified that leads us to form the opinion that fluid administration appears to confer an advantage, with added glucose proving even more beneficial. In both circumstances the mechanism is not clear but presumably is related to repletion of extracellular fluid and modification of the hormonal stress response. Perioperative fluid administration to all patients who have minor surgery warrants further examination since there appears to be some benefit in those who receive fluid.

Acknowledgments

The authors thank the staff of the day surgery ward and theatre suite at Churchill Hospital, Oxford, for their cooperation. We also thank Dr A. Mortimer for his assistance in setting up the trial and Ms H. Butler for her labours in preparing this manuscript.

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Blood contamination of anaesthetic and related staff

C. A. HARRISON, D. W. ROGERS AND M. ROSEN

Summary

The incidence of skin contamination of anaesthetic and related staff by patient's blood and saliva was studied during 270 anaesthetics in Cardiff hospitals over seven continuous days in October 1989. A survey was also made of current Hepatitis B immunisation status and glove-wearing habits of 75 anaesthetists. Blood from 35 (14%) patients caused skin contamination of 65 staff during 46 incidents. Twenty-eight (61%) of the contamination incidents occurred during vessel cannulation. Five (8%) of the 65 staff contaminated by blood already had cuts on their hands. There were nine incidents (4%) of skin contamination by saliva. Fifty-three (71%) anaesthetists were immunised against Hepatitis B. Only seven (9%) anaesthetists wear gloves for oral or nasal intubation, six (8%) for insertion of peripheral venous cannulae, 47 (63%) for insertion of arterial lines and 67 (89%) for insertion of central lines. All anaesthetic and associated staff should wear gloves on a routine basis.

Key words

Infection; Hepatitis B, acquired immune deficiency syndrome. Anaesthetists; complications.

The Association of Anaesthetists advises all anaesthetists to wear gloves in the operating theatre for vessel cannulation and insertion or removal of airways and tracheal tubes.¹ The Expert Advisory Group on the acquired immune deficiency syndrome (AIDS) also recommends gloves to be worn, as a minimum protective measure, for any procedures where there is likely to be contact with blood.² This advice was produced as a response to growing concern about the risk to operating theatre staff of infection from blood and secretions from patients with hepatitis or human immune deficiency virus (HIV) infection.¹⁻³

This study has three objectives: to determine, the incidence of skin contamination of anaesthetic and related staff by patient's blood and saliva during a normal working week, the current Hepatitis B immunisation status of anaesthetists in Cardiff and the incidence of glove usage.

Methods

A questionnaire which asked about skin contamination by blood and saliva was attached to each anaesthetic record of every patient who received an anaesthetic in the main operating theatres at University Hospital of Wales and Cardiff Royal Infirmary for a 7-day working week in October 1989. The type of operation, nature of surgery (elective or emergency), grade of anaesthetist and occur-

rence of skin contamination by blood or saliva were recorded.

Each anaesthetist in Cardiff was interviewed to determine whether he or she routinely wore gloves for oral or nasal tracheal intubation, the insertion of peripheral venous cannulae, arterial cannulae or central lines. Hepatitis B immunisation status was recorded. Comments were also sought as to the reasons why each did not routinely wear gloves in the operating theatre.

Results

Skin contamination by patients' blood and saliva

Two hundred and seventy anaesthetics were administered during the 7-day continuous period in the two hospitals, of which 252 (93%) forms were returned. Eighty-three percent (209) had elective surgery and 17% (43) emergency surgery. A total of 256 peripheral cannulae (some patients had more than one peripheral line while others had a venous cannula already *in situ*), 21 arterial lines and 20 central lines (nine drum catheters and 11 internal jugular lines) were inserted.

Blood from 14% (35) patients caused skin contamination of 65 people during 46 incidents (there was often more than one contamination incident per patient). Seventy-six percent (27) of the 35 patients involved in blood con-

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Accepted 28 March 1990.

Table 1. Causes of skin contamination.

Cause of contamination with blood	Number of incidents	Number of people contaminated			
		Anaesthetists	ODA/nurse	Other	Total
Vessel cannulation, peripheral	16	15	2	1	18
Central (drum catheter)	7	6	3	0	9
Arterial 'A'	5	10	4	0	14
Sampling from 'A' line	2	2	0	0	2
Needlestick injury	1	1	0	0	1
Attaching administration set to cannula	6	5	2	0	7
Stored blood	4	6	1	0	7
Traumatic tracheal intubation	1	1	0	0	1
Splashes from surgical field	4	2	2	2	6
Total	46	48	14	3	65

tamination were having elective surgery, and 24% (8) emergency surgery. Four percent (9) of incidents were caused by skin contamination by saliva. Table 1 lists causes of skin contamination by blood and the number of staff contaminated.

Sixty-one percent (28) of the contamination incidents were caused by vessel cannulation, 35% (16) during cannulation of a peripheral vein, 15% (7) by insertion of a central line into the antecubital fossa, and 11% (5) by insertion of an arterial line. There were also a further two incidents (4%) of contamination when samples were taken from an arterial line. Twenty-two percent (10) of incidents were caused by attaching transfusions to the peripheral cannula, six (13%) by patients' blood and four (9%) by donor blood whilst setting up a blood transfusion. Nine percent (4) of incidents of contamination were caused by blood splashes from the surgical field, one as an aortic cannula was removed and three from blood 'pooled' under the patient.

There was one needlestick injury (2% of incidents, 0.4% of all anaesthetics). One incident during a difficult intubation resulted in contamination by both blood and saliva of an anaesthetist's hands. There were no splashes of blood on mucous membranes or conjunctivae.

Of the 65 people contaminated by blood, 48 (74%) were anaesthetists, 14 (21%) were operating department assistants (ODAs) or anaesthetic nurses and three (5%) were other staff (one medical student, one trainee ODA and one monitoring technician). Five (8%) of the 65 staff contaminated had cuts on their hands at the time of blood contact with skin.

Current anaesthetic practice (Tables 2 and 3)

There are 75 anaesthetists in the anaesthetic department in Cardiff: 27 consultants, 14 senior registrars, 23 registrars including research registrars, and 11 senior house officers (SHOs). Only 9% (seven) of anaesthetists report wearing

gloves for oral or nasal intubation, and 8% (six) for insertion of peripheral venous cannulae. However 63% (47) wear gloves to insert arterial lines and 89% (67) for insertion of central lines (other than drum catheters) (Table 2). Fifty-three members (71%) of the department had been immunised against Hepatitis B.

The most common reason for not wearing gloves was loss of sensitivity and touch (42 replies), habit (25 replies), inconvenience (20 replies), discomfort (15 replies). Also cited were ignorance and laziness (10 replies), lack of protection against needlestick injuries or contamination by blood splashes on mucous membranes (seven replies). A few mentioned the low chance of infection from blood splashes to the skin (5 replies) and three mentioned difficulty in using adhesive tape, especially 'sleek' (Table 3).

Discussion

Theatre staff are regularly exposed to skin contamination by patients' body fluids. Concern about this relates to potential exposure of health staff to patients with unrecognised infection, especially Hepatitis B and HIV. Hepatitis B is highly infectious and only minute amounts of blood are required for transmission to occur. The incidence of the carrier state in Northern and Western Europe, North America and Australia is 0.1–0.5% of the population.¹ An anaesthetist who gives 1000 anaesthetics per year can therefore expect between 1 and 5 of his (her) patients per year to be Hepatitis B carriers, although this is likely to be an underestimate of the problem. Immunisation protects health care workers against Hepatitis B but as yet there is no protection against Hepatitis C or HIV.

Fortunately, most studies have shown that the risk of infection with HIV to health care workers is very low.^{5,6}

Table 3. Reasons for failure to wear gloves.

Reasons given	Replies
Loss of sensitivity and 'feel'	42
Habit	25
Convenience	20
Discomfort of wearing gloves	15
Ignorance and laziness	10
No protection against needle or eye contamination	7
Low chance of infection	5
Difficulty using adhesive tape to stick down (especially 'sleek')	3

Table 2. Glove-wearing habits.

	Yes	No
Oral/nasal tracheal intubation	7 (9%)	68 (91%)
Peripheral cannulation	6 (8%)	69 (92%)
Arterial cannulation	47 (63%)	28 (37%)
Central cannulation	67 (89%)	8 (11%)
Hepatitis B immunisation	53 (71%)	22 (29%)

The greatest risks for both Hepatitis B and HIV infection come from needlestick injuries, splashes to the mucous membranes, conjunctiva or nonintact skin.^{2,7} The wearing of gloves does not protect against the first three of these possibilities. Fortunately, in this study the incidence of needlestick injury was only one in 252 (0.4%) and there were no cases of splashes to the mucous membranes or conjunctivae. However, skin contamination with blood from HIV-infected patients has resulted in seroconversion in three health care workers.⁸ The interval between infection and antibody production is usually within the first 18 months, but may be as long as 42 months.⁹ Testing of every patient is no guarantee of safety even if it were ethically permissible. By the end of October 1989, 2717 cases of AIDS had been reported to the Public Health Laboratory Service AIDS centre.¹⁰ The number of HIV carriers is estimated to be 36 000–148 000.¹¹ An anaesthetist might therefore expect on average between one and three of his (her) patients per year to be HIV positive.

In this study, 14% of the anaesthetics administered gave rise to contamination incidents. Eight percent of those staff contaminated had cut skin, which is a surprisingly high figure. Peripheral venous cannulation is only relatively safe; 6% of insertions give rise to blood contamination of skin. There were no cases of contamination with blood during insertion of internal jugular lines because most anaesthetists wore gloves. However, most drum catheters inserted in the antecubital fossa resulted in blood contamination; the design is such that contamination is likely. Arterial line insertion is also a potentially high-risk area. A further 22% of incidents were caused by setting up intravenous infusions. Anaesthetists, and those assisting them, should clearly wear gloves routinely, if the incidence of contamination is to be cut down considerably when inserting arterial lines, central lines (especially drum catheters), and setting up intravenous infusions. Only 71% of anaesthetists in this department, despite personal appeals, are immunised against Hepatitis B and many are clearly still unaware of the risks of contamination with infected blood. This fact is supported in that only 8% of Cardiff anaesthetists wear gloves for peripheral venous cannulation and only 9% for intubation. The numbers who wear gloves for higher risk central and arterial cannulation increase to 89% and 63% instead of nearer 100%.

The commonest reason for not wearing gloves routinely is the loss of sensitivity. However, surgeons operate successfully with gloves as do most anaesthetists when they undertake local anaesthetic blocks. Dexterity whilst wearing gloves is a matter of practice which some have acquired. Many anaesthetists complained of the discomfort of wearing gloves for long periods. In that case gloves could be removed after induction of anaesthesia and donned again when necessary for managing 'blood' or at extubation. This would also prevent contaminating the apparatus with blood or saliva from gloves.

Most anaesthetists preferred to wear sterile surgical gloves rather than cheaper disposable gloves. Surgical gloves are readily available in all sizes and fit well. Surgeons require these qualities to carry out surgery. Anaesthetic procedures are often no less delicate. The use of poorly fitting gloves that do not provide the same degree of tactile sensitivity is a hindrance rather than a help.

This survey suggests that good quality gloves in a full range of sizes should be available in anaesthetic rooms. To emphasise the dangers of skin contamination by blood and other body fluids, each incident should be regarded as an accident and an entry made in an accident book. All anaesthetists and associated staff (ODAs nurses etc) should wear gloves on a routine basis. Junior anaesthetic staff should be encouraged at the beginning of their anaesthetic career to wear gloves so that the practice becomes routine.

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Attenuation of suxamethonium myalgias

Effect of midazolam and vecuronium

M. L. MINGUS, A. HERLICH AND J. B. EISENKRAFT

Summary

We studied the incidence of fasciculations and postoperative myalgias in 100 female outpatients who had laparoscopy under thiopentone, N₂O, isoflurane anaesthesia. Four groups of 20 patients each were pretreated with saline (group 1), tubocurarine 0.05 mg/kg (group 2), vecuronium 0.006 mg/kg (group 3), or midazolam 0.025 mg/kg (group 4), followed by suxamethonium 1.5 mg/kg. Group 5 received only vecuronium 0.1 mg/kg as relaxant (no suxamethonium). Fasciculations were graded, and postoperative myalgias rated on the first and third postoperative days. In groups 1–5 the incidence of fasciculations was 95, 15, 25, 95 and 0%; the incidence of myalgias on the first day after operation was 70, 45, 65, 75 and 60%, and on the third day after operation 20, 5, 20, 20, and 5%, respectively. We conclude that pretreatment with vecuronium, but not midazolam, decreases the incidence of fasciculations after suxamethonium ($p < 0.05$) and that in this patient population, postoperative myalgias appear to be unrelated to the use of suxamethonium.

Key words

Complications; fasciculations, myalgias.

Neuromuscular blocking agents; tubocurarine, suxamethonium, vecuronium.

Postoperative myalgias (POM) are common among patients who receive suxamethonium; the reported incidence ranges from 0.2–89%.^{1–7} Myalgias occur more frequently after minor operative procedures in women and in outpatients.^{1,2,4–7} Muscle fasciculations are also common after suxamethonium administration and have been associated with the development of POM.^{8,9} Various pretreatment regimens have been used in an attempt to decrease the incidence of fasciculations and POM. These include a small dose of a non-depolarising muscle relaxant,^{1,4,6,9,10–16} a benzodiazepine,^{17–20} lignocaine,^{21,22} and suxamethonium itself.^{23,24}

The purpose of this study was to determine whether pretreatment with a small dose of vecuronium or midazolam would attenuate fasciculations and (or) POM after suxamethonium in female patients who have laparoscopy as outpatients. The study was subsequently extended to determine whether myalgias after laparoscopy were associated with the use of suxamethonium.

Methods

We first studied 80 ASA physical status 1 and 2 female patients, after Institutional Review Board Ethics Commit-

tee approval and written informed consent was obtained. The patients had received no premedication and were scheduled to undergo laparoscopy on an outpatient basis. Each patient was assigned to one of four pretreatment groups, in a prospective, double-blind, randomised study to receive: group 1, saline; group 2, tubocurarine 0.05 mg/kg; group 3, vecuronium 0.006 mg/kg; and group 4, midazolam 0.025 mg/kg. All patients were monitored with ECG, noninvasive blood pressure (Dinamap, Critikon Inc., Tampa, FL, USA), pulse oximetry, mass spectrometry, and an oesophageal temperature probe. Fentanyl 100 µg and droperidol 1.25 mg were administered intravenously after insertion of an intravenous cannula, followed, at time zero, by the pretreatment drug according to group. Anaesthesia was induced 1.75 minutes later with sodium thiopentone 4 mg/kg. Suxamethonium 1.5 mg/kg was given at 3 minutes to facilitate tracheal intubation. An observer, blinded to the patient's pretreatment, then rated the fasciculations on a scale of 0–3, where 0 = none; 1 = mild fasciculations of eyes, face, neck or fingers without limb movement; 2 = moderate fasciculations involving limbs and/or trunk; and 3 = severe vigorous motion requiring restraint of limbs.^{14,15} Anaesthesia was maintained with N₂O (60%) in O₂ and isoflurane 0.5–1.5% end-tidal concentration as measured by mass spectrometry (Perkin-Elmer Advantage 1100,

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Accepted 22 February 1990.

Pomona, CA, USA). None of the patients in groups 1–4 required additional muscle relaxant.

A nurse investigator, blinded to the pretreatment group, interviewed each patient by telephone and questioned them about POM on the first and third postoperative day. Postoperative myalgias were scored on a scale of 0–3; where 0 = none, or absence of pain other than characteristic post-laparoscopic gas pains; 1 = mild muscle stiffness in the neck or shoulders; 2 = moderate muscle stiffness requiring analgesics; and 3 = severe incapacitating generalised muscle stiffness or pain.^{14,15} A fifth group of 20 ASA 1 or 2 female outpatients, also scheduled to undergo laparoscopy, was added and studied after ethics committee approval and written informed consent was obtained, and after completion of the study of 80 patients (groups 1–4), and analysis of the data. In this group (group 5), the anaesthetic management was as described previously with the exception that neuromuscular block was achieved with vecuronium 0.1 mg/kg (i.e. no patient received a pretreatment drug or suxamethonium). Fasciculations and POM were assessed as previously described, by a blinded nurse investigator. Data were analysed by analysis of variance, Chi-squared, and contingency coefficient analysis. A value of $p < 0.05$ was considered significant.

Results

We found no significant difference in mean age or weight among any of the five study groups (Table 1). The results of the Chi-squared analyses for fasciculations are presented as percentages in Table 2. We also grouped together, for ease of comparison, none with slight fasciculations (scores 0 or 1), and moderate with severe fasciculations (scores 2 or 3) (Table 2). We also analysed none compared with any fasciculations (scores 0 compared with 1, 2, or 3) (Table 2) and found no difference in the incidence of fasciculations among groups 2, 4 and 5, or between groups 1 and 3. The incidence of fasciculations was significantly greater in groups 1 and 4, than in groups 2, 3 and 5 ($p = 0.000$). We found no significant differences in the severity of fasciculations between groups 1 and 4, or between groups 2, 3 and 5. We found no associations among the pretreatment groups between the severity of fasciculations and POM. None of the patients demonstrated severe fasciculations (score of 3).

The results of the analyses for POM on the first day after operation are shown in Table 3. There were no significant differences among any of the five groups; the overall incidence (for the 100 patients) was 61%. The mean incidence of POM (for the 100 patients) by the third day after operation had decreased to 14% (Table 4). We found no statistically significant differences among the groups when myalgia categories were compared as none versus any, or none/slight versus moderate/severe on either the first or

Table 1. Demographic details; mean (SD).

Group	Age (years)	Weight (kg)
1 Saline pretreatment	34 (4)	60 (12)
2 Tubocurarine pretreatment	34 (5)	59 (10)
3 Vecuronium pretreatment	33 (6)	61 (9)
4 Midazolam pretreatment	35 (4)	58 (6)
5 Vecuronium alone (no suxamethonium)	34 (6)	62 (10)

Table 2. Incidence of fasciculations (%). $n = 20$ in each group.

Group	Fasciculation score			
	0	1	2	3
1	5	25	70*	0
2	85	15	0	0
3	75	15	15†	0
4	5	10	80	0
5	100	0	0	0
	None/slight (score 0, 1)		Moderate/severe (score 2, 3)	
Group				
1	30		70*	
2	100		0	
3	85		15†	
4	20		80	
5	100		0	
	None (score 0)		Any (score 1, 2, 3)	
Group				
1	5		95*	
2	85		15	
3	75		25†	
4	5		95	
5	100		0	

* $p < 0.05$: group 1 compared with group 5.

† $p < 0.05$: group 4 compared with group 5.

Table 3. Incidence of myalgias (%) on the first day after operation. $n = 20$ in each group.

Score	0	1	2	3
Group				
1	30	60	10	0
2	55	40	5	0
3	35	45	10	10
4	25	45	20	10
5	40	50	5	5
	None/slight (score 0, 1)		Moderate/severe (score 2, 3)	
Group				
1	90		10	
2	95		5	
3	80		20	
4	70		30	
5	90		10	
	None (score 0)		Any (scores 1, 2, or 3)	
Group				
1	30		70	
2	55		45	
3	35		65	
4	25		75	
5	40		60	

All between-group comparisons, $p > 0.05$.

third day after operation. We found no association between the presence of fasciculations and the presence of myalgias.

Discussion

Suxamethonium remains one of the most useful drugs in anaesthesia. It is inexpensive and has a rapid onset and

Table 4. Incidence of myalgias (%) on the third day after operation. $n = 20$ in each group.

	None/slight (score 0, 1)	Moderate/severe (score 2, 3)
<i>Group</i>		
1	100	0
2	100	0
3	95	5
4	95	5
5	100	0
	None (score 0)	Any (scores 1, 2, or 3)
<i>Group</i>		
1	80	20
2	95	5
3	80	20
4	80	20
5	95	5

All between group comparisons, $p > 0.05$.

short duration of action, no tissue toxicity, and a relative lack of cardiovascular side effects. Disadvantages include fasciculations and postoperative myalgias, which may be more common among women and outpatients.²⁵ Various 'pretreatment' drugs have been used in an attempt to prevent side effects; these include, tubocurarine^{1,4,9} other non-depolarising muscle relaxants,^{6,10-16} benzodiazepines,¹⁷⁻²⁰ lignocaine,^{21,22} and suxamethonium itself.^{23,24} The reported incidence of fasciculations varies among the different studies, but in general, in the nonpretreated groups, ranges between 90 and 100%.^{3,9,15} Female patients were chosen for our study because they are reported to show a higher incidence of POM when compared with males.¹⁻⁴

The 95% incidence of fasciculations in group 1 (saline) is comparable with the results of other studies.^{3,9,15} Erkola *et al.*¹³ reported that pretreatment with tubocurarine 0.05 mg/kg, or vecuronium 0.01 mg/kg, given 3 minutes before suxamethonium 1.5 mg/kg, decreased the incidence of fasciculations from 95% in their control group to 24 and 54%, respectively.¹³ The 95% incidence of fasciculations in our control group (group 1) was reduced to 15% in group 2 tubocurarine and 25% in group 3 (vecuronium), with comparable pretreatment doses of tubocurarine or vecuronium.

Several studies have reported that pretreatment with diazepam 0.05 mg/kg can decrease fasciculations and POM after suxamethonium.^{17,18} Others, however, using similar doses of diazepam, have been unable to demonstrate any significant reduction in fasciculations or POM after suxamethonium.²⁷ The incidence of fasciculations in our study in group 4 (midazolam) was 95%, which was not significantly different from group 1 (saline). We chose a midazolam dose of 0.025 mg/kg because it is reported to have two to three times the potency of diazepam in its other properties.²⁸

Most studies to date have failed to demonstrate a clear association between fasciculations and POM.^{5,14,15,21} Sosis *et al.*¹⁴ studied pretreatments with atracurium 0.025 mg/kg and tubocurarine 0.05 mg/kg and found that, while tubocurarine was better at preventing fasciculations (88% compared with 54%), atracurium was better at preventing POM (85% compared with 59%). Many other studies have

shown that, while non-depolarising relaxants may decrease POM after suxamethonium, they do not abolish them completely.^{1-5,10} One study compared three anaesthetic techniques using isoflurane, with or without suxamethonium, in outpatients undergoing laparoscopy.³⁰ They found no difference in the incidence of POM among patients who received isoflurane: by mask without suxamethonium, by tracheal tube after precurarisation with tubocurarine followed by suxamethonium, or by tracheal tube after precurarisation with tubocurarine followed by suxamethonium and a suxamethonium infusion.³⁰ Zahl *et al.*²⁹ also studied the incidence of POM among women after outpatient laparoscopy. They substituted vecuronium 0.05 mg/kg for suxamethonium, and, even though vecuronium was not associated with fasciculations, the incidence of myalgias on postoperative days 1 and 2 was no different from those in the groups which had received suxamethonium.

We found no difference among groups 1-4 in the incidence of POM on either the first or third days after operation. Use of vecuronium 0.006 mg/kg as a pretreatment drug significantly reduced the incidence of fasciculations, but not the incidence of POM on either the first or third days after operation. In contrast to the Sosis *et al.*¹⁴ study, none of our pretreatment drugs was more effective than any other in preventing POM, despite significant differences among the pretreatment groups in the incidence of fasciculations.

To investigate further the possible association between suxamethonium and POM, we subsequently studied a group of 20 patients to whom vecuronium alone was given to produce neuromuscular blockade (group 5). None of the patients in group 5 had fasciculations and, as before, we failed to find a significant reduction in the incidence of myalgias on either the first or third day after operation. Indeed, we found no difference between the incidence of myalgias on the first and third days after operation in group 5, and those in groups 1-4 who had received suxamethonium.

Assignment to group 5 was not random in this study and was performed after completion of the study of groups 1-4, but the mean ages and weights of group 5 patients did not differ significantly from those in groups 1-4. In addition, the nurse observer who graded the myalgias in group 5 was unaware of the treatment given. We therefore believe that the comparison of results from group 5 with those from groups 1-4 is valid.

Our results support the findings of Zahl *et al.*²⁹ who found that, in the female patient who had outpatient laparoscopy, POM bore no relation to the administration of suxamethonium, with or without pretreatment, or to fasciculations. Perhaps in this particular group of patients, POM are related more to the degree of surgical manipulation and to the dissection of tissues by CO₂ gas, all of which can contribute to shoulder pain and backaches, than to the muscle relaxant technique used. This type of patient may have too much generalised discomfort from gas pains, which may mask any POM arising from suxamethonium. Studies need to be performed on patients who have surgical procedures other than laparoscopy in order to explore further this possibility.

It was interesting that midazolam appeared to be ineffective in attenuating either fasciculations or POM, in contrast to the results of previous studies using diazepam.¹⁷⁻²⁰ Laurence *et al.*²¹ found that a pretreatment dose of midazolam

0.15 mg/kg failed to decrease the incidence of fasciculations and myalgias on the first day after operation following suxamethonium given as a bolus dose of 1.2 mg/kg followed by incremental doses. It has been hypothesised that diazepam may act centrally at the level of the spinal cord to exert its muscle relaxant effects.¹⁹ Both are benzodiazepines, although perhaps midazolam does not share this property of diazepam.

We conclude that, apart from preventing aesthetically displeasing fasciculations, there seems to be no advantage in pretreating the outpatient female laparoscopic surgical patient before administration of suxamethonium. This patient population would appear to be a poor model for the study of postoperative myalgias in relation to neuromuscular block technique.

Acknowledgments

The authors are grateful to J. Victor Reyniak, MD for his cooperation and to Mrs B. Perez-Rivera for her administrative support.

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Clinical presentation of suspected malignant hyperthermia during anaesthesia in 402 probands

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Summary

As anaesthetists have become more aware of malignant hyperthermia the mortality rate has fallen, but concomitantly the number of dubious and aborted cases has increased. All probands who developed a suspected malignant hyperthermia reaction during anaesthesia and subsequently underwent muscle biopsy were classified according to the clinical presentation. A probability for malignant hyperthermia can be calculated, using the classification, for each type of clinical presentation; this varied from 0.96 to 0.07. Certain clinical features were found to be of more value as predictors than others; these included a high creatine kinase and myoglobinuria. The accuracy of prediction depends on a clear contemporaneous description of the clinical events.

Key words

Hyperthermia; malignant.

The fulminant, classical occurrence of a malignant hyperthermia (MH) reaction is now encountered less frequently because of increased awareness of the condition by anaesthetists, and better use of monitoring facilities. There is also an increase in the number of aborted cases, in which anaesthesia is stopped and treatment instituted as soon as MH is thought to be likely. This means there are fewer obvious signs of MH and, since no clinical sign is unique to MH, the decision as to whether to proceed to further investigation is made more difficult. Not only has the number of aborted cases of MH increased but also the overall number of cases referred to us. In this paper, by relating the type of clinical presentation of MH with the results of *in vitro* muscle biopsy screening, we hope to identify which features of the clinical presentation carry a higher probability for an accurate clinical diagnosis of MH.

There is no current method of classifying the clinical reactions of MH. Britt¹ grouped patients as having rigid or nonrigid reactions, but this has not been adhered to by others. The attempt to categorise clinical reactions naturally followed the recognition of masseter spasm as a clinical problem. The classification used in this paper was developed from an original suggestion by Ørding and Ranklev (personal communication).

Methods

Classification of the probands

All probands referred to the unit after a suspected MH reaction during anaesthesia and who were investigated by

muscle biopsy are included. If the proband had died or was too young to be investigated, they were 'diagnosed' indirectly using the results of relatives, usually parents. There was no complete record of all the signs associated with MH in the majority of cases, in particular the recognition and measurement of myoglobinuria. However, the clinical history often gave a good 'impression' of the problem and the patient was categorised accordingly. It was therefore impossible to keep to strict objective criteria when classifying each patient. Each proband was allocated to one of eight mutually exclusive categories, according to their clinical history given by the referring physician, as follows.

Fulminant/classical. Marked evidence of both metabolic stimulation and abnormal muscle activity; the signs include metabolic acidosis, hyperthermia ($> 38.5^{\circ}\text{C}$), arrhythmias, hyperkalaemia, muscle rigidity, myoglobinuria and greatly raised creatine kinase (CK) (> 1500 IU/litre). These probands all received active treatment and the situation appeared life-threatening (category *a*).

Moderate. Inconclusive signs of MH that involved both metabolic and muscle anomalies with MH being a probable diagnosis. These patients either did not receive active treatment or only a single dose of dantrolene. No treatment was required after withdrawal of trigger agents and the situation never appeared life-threatening (category *b*).

Mild. Signs only of a metabolic derangement such that MH is a possible diagnosis, but with a pH > 7.3 and a body core temperature never more than 38.5°C (category *c*).

Masseter spasm with evidence of rhabdomyolysis, i.e. with

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Accepted 12 October 1989.

a CK of > 1500 and myoglobinuria (category *d*).

Masseter spasm with signs of metabolic disturbance (e.g. arrhythmia, rising core temperature) (category *e*).

Masseter spasm only (category *f*).

Unexplained peri-operative death or cardiac arrest (category *g*).

Others. Postoperative pyrexia, postoperative rhabdomyolysis (category *h*).

Patients were grouped according to the year of biopsy, because of differences in muscle biopsy techniques and interpretation of results of the *in vitro* contracture studies. The first group, 1971–1976, represents those patients in whom any sustained muscle contracture with 2% halothane or less was taken to indicate MH susceptibility (MHS). The second group, 1977–1981, represents a period of established *in vitro* tests, using both halothane and caffeine independently as stressors, but before the formation of the European MH Group (EMHG). The final group, 1982–1987, includes all those probands tested according to the European protocol.^{2,3} All the probability values quoted in the results section refer to this period only.

Laboratory screening interpretation

The laboratory investigation of MH results in patients being categorised as susceptible to MH, when both the halothane and caffeine tests are abnormal, or unsusceptible to MH (MHN) when neither test is abnormal. However, since 1984–85 when the EMMG protocol was adopted, a third category was added, MHE, which is used when either the halothane or the caffeine challenge test is abnormal but not both. For the purpose of this study all patients shown to be MHE were included in the MHS group since all MHE probands must be regarded as susceptible to MH once another family member has been shown to be MHS.

Not all probands referred to the unit are investigated by muscle biopsy and obviously cannot be included in this study. These patients usually fall into three groups; firstly those in whom there is another reasonable explanation e.g. concurrent infection with pyrexia and a high white cell count; secondly a probable normal reaction to anaesthesia and surgery e.g. mild postoperative pyrexia in a child; and thirdly an artefactual case displaying signs of MH e.g. the consequences of an increased end-tidal CO_2 with a Bain's system, or another muscle disorder such as myotonic dystrophy proved subsequently by EMG.

Results

Altogether 402 probands were investigated over the whole period 1971–1987. Two hundred and sixteen probands (54%) were shown to be MHS and 186 (46%) MHN. The total number of patients has greatly increased since the

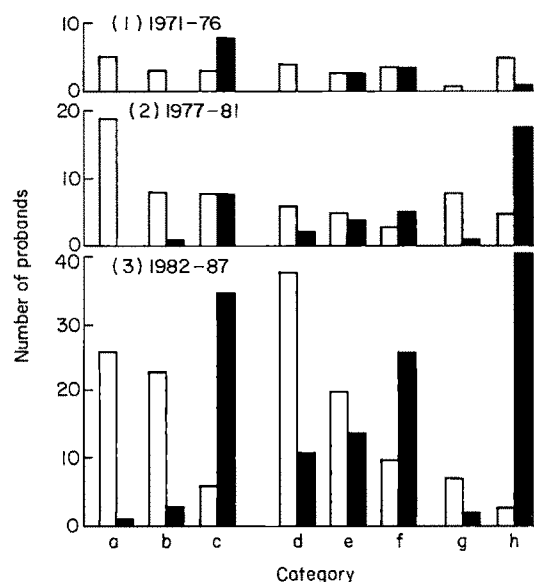


Fig. 1. Distribution of the probands in the eight categories over the time periods 1971–6, 1977–81 and 1982–87. □ MHS, ■ MHN.

inception of the unit; in each consecutive time period the numbers of probands are 40, 93 and 269, but with a decreasing percentage of probands found to be MHS, 60%, 63% and 49% consecutively.

The distribution of the probands throughout the eight categories over the three time periods is shown in Figure 1. For the last time period only (1981–87), the probability of the prediction of MH for each category is shown in Table 1.

Category a. The pattern over the three time periods is unaltered. In the first two periods all probands in this category were MHS; however, in the latest period 1982–87, one proband was MHN on laboratory testing. The probability of being MHS in this group is 0.96.

Case 1 was a boy aged 16 years. There are doubts about the true parentage, which makes the diagnosis of an inherited disease impossible.

Category b. This shows an unchanging pattern over the periods considered, but the proportion of MHN patients, although remaining small, has increased; there are three in the final period, so that the probability of being MHS is 0.88.

Cases 2 and 3 were remarkably similar. Both probands were teenage boys who required surgery after motorbike accidents and developed signs of carbon dioxide retention, arrhythmias and acidosis with spontaneous respiration via a Bain's system (in our experience a frequent problem). Elevated CK and myoglobinuria were found but could have been from the extensive muscle damage caused by the

Table 1.

Clinical category	Incidence of MHS
(a) Severe	0.96
(b) Moderate MH	0.88
(c) Mild	0.14
(d) Masseter spasm with muscle involvement	0.76
(e) Masseter spasm with metabolic signs	0.57
(f) Masseter spasm alone	0.28
(g) Unexplained anaesthetic death/cardiac arrest	0.66
(h) Others, postoperative pyrexias	0.07

original trauma. Case 4 was classified as moderate because the temperature and pH decreased outside the limits for the mild group.

Category c. The pattern for the middle period differs from the other two. In the final period there is a great increase in the numbers who present in this category; the majority are MHN. The probability of a mild clinical presentation from MHS is 0.14. Of the five patients in this category who were shown to be MHS there was no clinical feature which clearly indicated a potential MH-susceptible case and no case had all the laboratory investigations performed. Evidence of increasing CO₂ production and CK were the most useful features. One case, with hindsight, might have been allocated to category *e* because of 'some difficulty' in mouth opening. The referring anaesthetist in all these cases was obviously very concerned about the patients' reactions.

Categories d, e, f. These concentrate on clinical presentations where masseteric spasm is a marked feature. Throughout the three time periods, with increasing numbers in these categories a clearer pattern emerges. In the final period, 1982–87, masseter spasm with evidence of muscle involvement (*d*) carries a 0.76 probability of MHS. When it occurs with other signs (*e*) this is reduced to 0.59 and when it occurs alone (*f*) the probability is 0.28 (Table 1).

Category g. All three time periods show a similar pattern, with 66% of probands being MHS.

Category h. The pattern for this category is the same for all time periods but is more accentuated in the latest period, where the probability of being MHS is only 0.07. The type of case referred included postoperative pyrexia (the largest numerical group $n = 32$), postoperative rhabdomyolysis, postoperative renal failure, possible significant family history, and 'trouble' or 'reactions' with anaesthesia. Thirty-one of the 32 probands who presented with a postoperative pyrexia were shown to be MHN, an observation found by other Units. The only case which proved to be positive had been specifically told he was 'allergic to halothane'.

All the deaths from MH are found in category *a*; the rates are 100%, 53% and 54% in the three time periods respectively. The improvement in mortality, currently 2–3%, is only very recent.⁴ There were no deaths associated with the categories that involve masseter spasm (*d, e, f*). Five apparently normal fit patients who presented in these groups were found to have myotonia congenita (confirmed histologically and by EMG studies) but were not MHS according to the *in vitro* tests.

Discussion

It is reassuring to find that nearly all the probands presenting with fulminant (*a*) or moderate (*b*) MH reactions were found to be MHS on laboratory testing. However, there were four probands from these two categories who were found to be MHN, which vindicated our stance for biopsying all probands regardless of the clinical history; it should be emphasised that all the 'signs' of MH are nonspecific.

The change in pattern seen in category *c* may be due to several factors. Firstly, the number of probands is quite small in the earlier compared to the later periods; secondly, the quality of referral information improved; thirdly, there

was increased awareness by the anaesthetist of the possibility of an MH reaction, which would be expected to increase the number of potential MHN probands. Some of the early referrals are extremely well documented, although poorly documented cases were more frequent than latterly, making incorrect classification more likely. Surprisingly, category *g* showed the same pattern throughout, but with a slight decrease in the number of patients who were MHS. This stresses the importance of assessing pre-operatively the family's anaesthetic experience.

The findings in category *h* support our view that postoperative pyrexia alone is unlikely to be MH. Postanaesthetic renal failure secondary to rhabdomyolysis may also be due to MH but could reflect other muscle disorders. Histological examination of the muscle plays an important role in these cases.

It may be that better record-keeping would have helped in those cases classified in groups *c, f, g*, and *h* and who were found to be MHS. Better record-keeping would include a timed sequence of clinical events, together with laboratory results. The clinical timed-events that are important are: masseter spasm (duration and degree), generalised muscle rigidity, arrhythmias, tachypnoea and (or) signs of increased CO₂ production, cyanosis and increase in body core temperature. The laboratory investigations should always include arterial blood gases, serum potassium levels, initial 12- and 24-hour CK measurements and evidence of myoglobinuria; the two latter measurements are extremely useful in the probands whose main clinical problem is masseter spasm. A description of the clinical events can also help in the interpretation and understanding of the data.

For the future it may be possible to predict MH using a variety of tests, each of which is only of limited predictive value. The clinical presentation could be combined with a battery of, as yet, unconfirmed noninvasive laboratory tests such as MRI spectroscopy,^{5,6} membrane fluidity,⁷ intracellular calcium flux measurements,^{8,9} EMG,¹⁰ and muscle relaxation rates.^{11,12} Patients could then be allocated into one of three categories, MHS, MHN, and doubtful, and only the latter group would need further investigation by muscle biopsy, which at present remains the only reliable means of diagnosis.

Acknowledgment

A summary of this paper was included in a presentation of aspects of MH at the Association of Anaesthetists' meeting in Swansea 1989.

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Treatment of atelectasis of upper lung lobes

Selective bronchial suctioning with J-shaped catheter tip and guide mark

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Summary

We developed a technique for blind bronchial suction using a curved-tip catheter with a guide mark, for the treatment of atelectasis of the lower and middle lobes of the lung. Suction of the upper lobe bronchi could not be performed because of the combination of the peculiar anatomy of the upper lobe bronchi with catheter design. We treated successfully two cases of atelectasis of the right upper lobes using a Rusch Metras bronchography catheter with a guide mark which is not readily available. Therefore we devised a J-shape tipped catheter with a guide mark. We have successfully treated 13 episodes of atelectasis of the right upper lobe in 10 patients and one episode in the left upper lobe in one patient with this new catheter.

Key words

*Airway; selective bronchial suction.
Equipment; suction catheters.*

Atelectasis is a common postoperative pulmonary complication. Physicians skilled in fiberoptic bronchoscopy have used the technique extensively in its treatment, but it is not always available. We therefore developed our own technique for selective bronchial suction using a curved-tipped catheter with a guide mark¹⁻⁵ and have (unpublished observation) successfully treated atelectasis of the middle or lower lobes in this way.

However, we found suction of the right upper and left upper lobe bronchus difficult using a commercially available curved-tipped catheter because of the peculiar anatomy of these bronchi and the form of the curved tip. A Rusch Metras bronchographic catheter with guide mark was then used successfully for the treatment of atelectasis of the right upper lobe, although this brand of catheter is not readily available. Consequently, we devised a J-shaped tip catheter with guide mark and we describe its successful use in the blind catheterisation of the right upper lobe and left upper lobe bronchi and the successful treatment of atelectasis.

Methods

A catheter can be easily constructed by hand using a pocket cigarette lighter and a guide mark made with a felt-tipped pen. The J-shaped catheters were formed from commercially available straight- or curved-tip catheters.

Insertion of the J-tipped catheter into the right upper lobe bronchus was made by measuring the distance from incisor teeth to carina and from carina to the right upper lobe bronchus by means of auscultation, as previously described.³ The tip of the tracheal tube was placed 3 to 5 cm above the carina;³ all measurements were recorded on the anaesthesia chart. The patient's head was placed in midposition in order to prevent deviation of the tip of the tracheal tube in the trachea.⁵ After the tracheal tube was placed and maintained at the centre of the mouth, the catheter was directed towards the right for suction of the right upper lobe. Resistance was encountered while the J-shaped tip of the catheter passed through the tracheal tube. The catheter was then advanced until that part of the

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Accepted 11 January 1990.

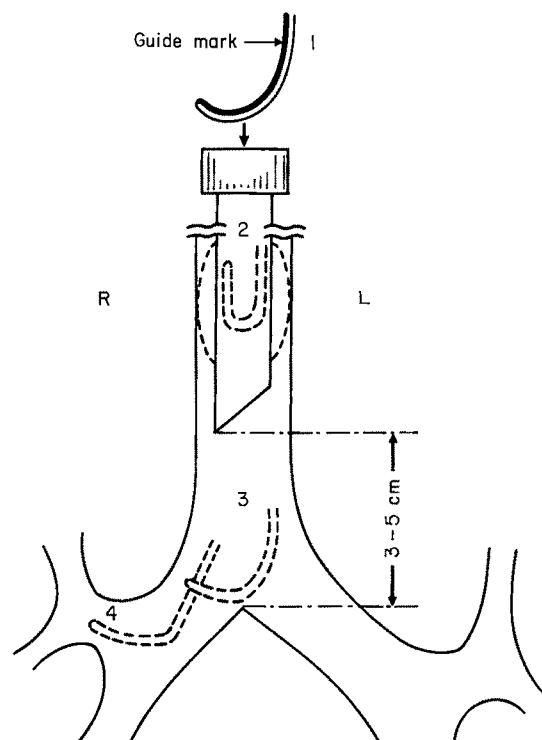


Fig. 1. Catheterising J-shaped tipped catheters into the eparterial bronchus.

catheter touched the carina, or at a predetermined distance. A cough was elicited at this point in patients not completely curarised. The catheter was then advanced (2–3 cm) until a second resistance was encountered. This indicated that the tip of the catheter was at the entry to upper lobe bronchus (Fig. 1).

The guide mark was directed towards the right during the entire procedure. A less acutely angled J-shaped tipped catheter than used for the right was employed for suction of the left upper lobe bronchus, which was advanced 4–5 cm beyond the carina.

Case histories

Case 1. A 51-year-old woman underwent emergency gastrectomy for gastric bleeding under general anaesthesia (Portex tracheal tube 7.5 mm; estimated distance from incisors to carina 25 cm; length of tracheal tube fixation 21 cm).

Atelectasis of the right upper lobe developed on the first postoperative day. External compression of the trachea, and transtracheal injection of saline were performed without improvement. Diazepam 10 mg was given intravenously and the trachea intubated with a Portex 7.5 mm tube and fixed at 21 cm. Several attempts at selective suction using a Portex 14-FG curved tip catheter with guide mark were performed without success. Blind selective suction of the right upper lobe using an acutely curved Rusch Metras bronchographic catheter (cat. No. 16400) with guide mark was performed three times. Copious secretions were recovered and atelectasis resolved completely (Fig. 2a, b).

Case 2. A 61-year-old man with a history of a ruptured left diaphragm from an aviation accident and subsequent empyema, underwent resection of a recurrent brain tumour and placement of an Ommaya catheter under general anaesthesia (tracheal tube: Bivona spiral 9 mm; incisors–carina: 29 cm; tube fixation: 24 cm). The chest X ray at the end of the operation revealed atelectasis of the left upper lobe. Several attempts at selective suction using a Portex 14 FG curved-tip marked catheter were performed without resolution of the atelectasis. Subsequent selective suction using a Portex 14 FG modified as described was performed twice and copious secretions were removed with the disappearance of atelectasis. Additional case reports are detailed in Table 1 and Figure 3.

Discussion

Atelectasis is a major complication after surgical procedures or during ventilatory care in the ICU. Pneumonia and rarely, a lung abscess may develop if untreated. The majority of instances of atelectasis can be treated by

Table 1. Successful treatment of atelectasis of the right upper lobes.

Number	Age	Sex	Anaesthesia	Operation	Onset of atelectasis	Treatment and size of catheter
3	18	M	None	None	First day of admission	SBS, Rusch Broncho*
					Second day of admission	SBS.JTCGM (14 FG)*
					Fourth day of admission	SBS.JTCGM (14 FG)*
4	12	M	General	Laminectomy L ₅ -S ₁	At the end of operation	SBS.JTCGM (14 FG)†
5	26	F	General	Removal of pancreatic cyst	At the end of operation	SBS.JTCGM (14 FG)†
6	7	M	General	Surgery of right hand	At the end of operation	SBS.JTCGM (10 FG)†
7	52	M	General	Gastrectomy for cancer	Second postoperative day	SBS.JTCGM (14 FG)†
8	62	M	General	Laminectomy C ₂₋₇	Seventh postoperative day	SBS.JTCGM (14 FG)*
					Eighth postoperative day	SBS.JTCGM (14 FG)*
9	46	M	General	Removal of brain tumour	300th postoperative day	SBS.JTCGM (14 FG)*
10	71	M	General	Gastrectomy for perforation of stomach	Second postoperative day	SBS.JTCGM (14 FG)†
11	59	M	General	Total pancreatectomy for cancer of pancreas	Sixth postoperative day	SBS.JTCGM (14 FG)†

SBS, selective bronchial suctioning; SBS.JTCGM, selective bronchial suctioning using J-shaped tipped catheter with a guide mark; † indicates that suction was performed through a tracheal tube and * indicates that suction was performed through a tracheostomy tube.

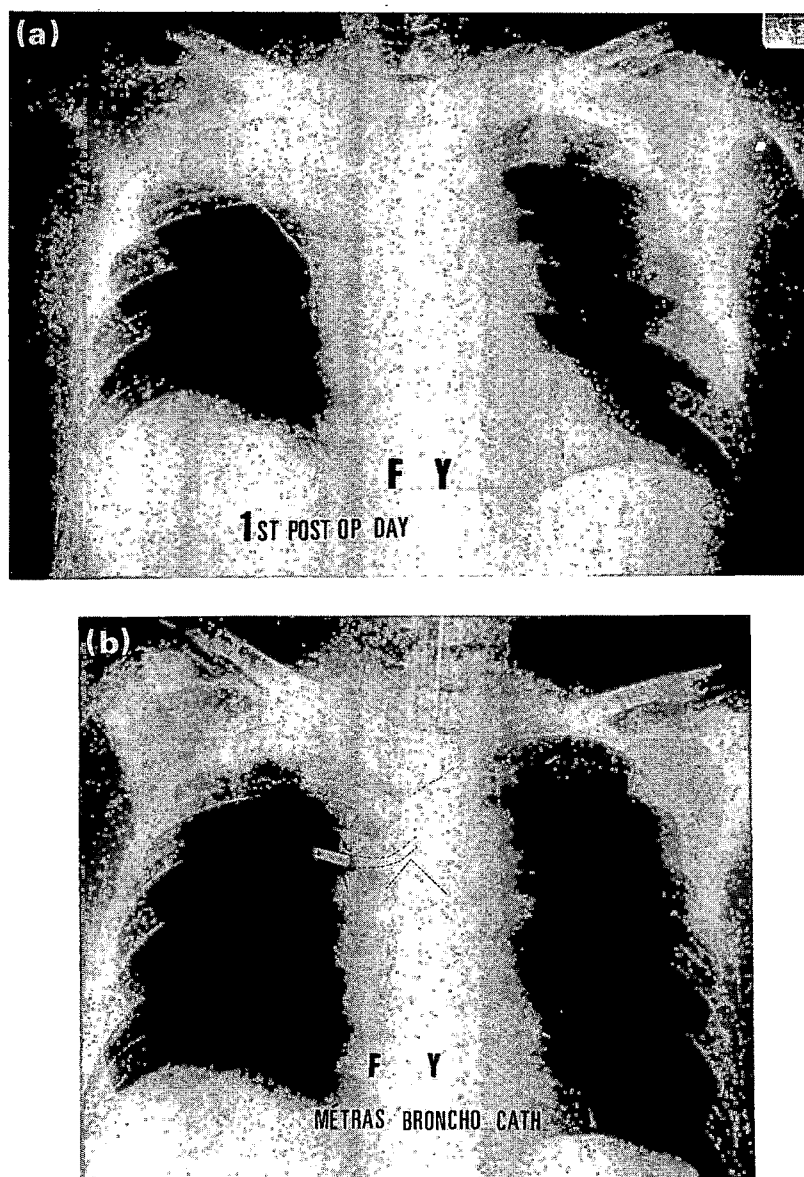


Fig. 2. Chest X ray (a) showing atelectasis of the right upper lobe in case 1 and (b) the tip of the Rusch Metras bronchography catheter in the eparterial bronchus after atelectasis had completely resolved.

conventional chest physiotherapy, external tracheal compression, and (or) instillation of saline or a trans-tracheal injection of a mucolytic agent, followed by suction down the trachea using a straight-tipped catheter. Fibreoptic bronchoscopy has been used extensively when atelectasis cannot be corrected by this means, but a fibreoptic bronchoscope and bronchoscopist may not be immediately available. We developed a method for selective suction using a curved-tipped catheter and have successfully treated atelectasis of the middle and lower lobes. However, when we found that atelectasis of the right upper lobe could not be treated successfully using this method, we devised a J-shaped tipped catheter with guide mark. Using this, we have successfully treated atelectasis of the right upper lobe. Catheterisation of the left upper lobe bronchus is also difficult, and the technique has been used to treat successfully atelectasis of the left upper lobe.

Sen and Walsh⁶ recently suggested that unnecessary fibreoptic bronchoscopy is employed in the treatment of atelectasis. Marini, Pierson *et al.*⁷ demonstrated that no significant difference existed in restoration of postoperative lung volume between bronchoscopy and chest physiotherapy. However, we have encountered many lobar atelectases which could not resolve by chest physiotherapy, tracheal compression, instillation of saline or mucolytic agent and tracheal suction using a straight catheter. The cases cited here could not be treated by the above techniques nor could atelectasis of the right upper lobe by selective suction. Therefore, we instituted selective suction using a J-tipped catheter. There has been no previous report of successful treatment of atelectases of the right and left upper lobe, as in our series, using only a suction catheter. We have not used fibreoptic bronchoscopy in the last 10 years to treat various types of atelectasis.

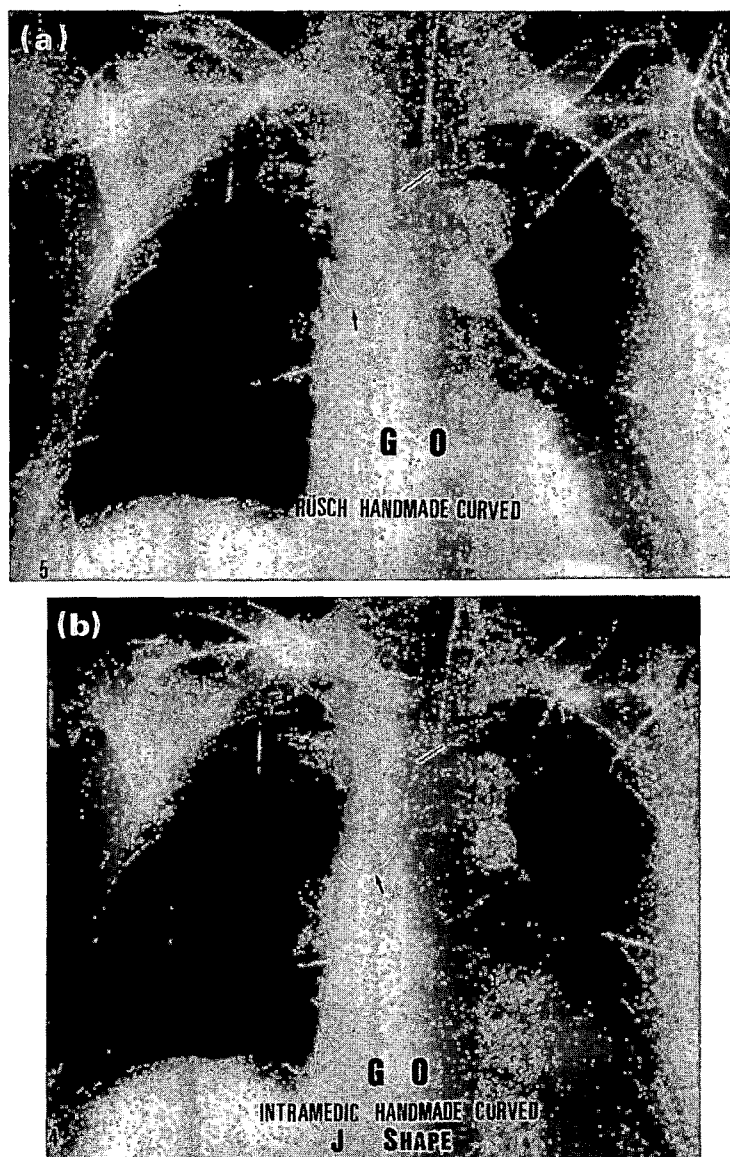


Fig. 3. Chest X ray (a) and (b) showing tips of J-shaped catheters placed in the right upper lobe bronchus after atelectasis treated with a J-shaped catheter (Portex 14 FG) in case 2.

Acknowledgments

The authors express their thanks to Dr L.D. Vandam of Harvard Medical School and Brigham and Women's Hospital for his comment and review.

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CASE REPORT

Spinal catheter anaesthesia for Caesarean section in a patient with spina bifida

F. NUYTEN AND M. GIELEN

Summary

A patient with grossly deformed vertebral anatomy, scheduled for elective Caesarean section, expressed her wish to stay fully awake during the procedure. Epidural anaesthesia was considered to be impracticable, while dural puncture appeared possible only at thoracic level. Spinal anaesthesia using a subarachnoid catheter placed at T₇₋₈ was employed successfully.

Key words

*Anaesthetic techniques, regional; spinal.
Complications; spina bifida.*

In patients with severe anomalies of the vertebral column, central conductance blockade may be rather difficult to establish.^{1,2} General anaesthesia is then frequently used. However, when a patient has a strong motivation to stay fully conscious during surgery, as is often the case for a Caesarean section, careful consideration of regional anaesthetic techniques is indicated. We report such a patient here.

Case history

A 33-year-old gravida 3 was admitted for elective Caesarean section because of cephalopelvic disproportion and neurological impairment of the lower body. She was born with a lumbosacral spina bifida aperta, which was surgically closed soon after birth. She was paraplegic, with hypoplastic deformed legs. Sensory disturbances existed in the lower lumbar and sacral segments. At the age of 19 years, a neuropathic bladder and bilateral hydronephrosis necessitated ureteral diversion by an ileal conduit. Mild normal pressure hydrocephalus existed since childhood, but intellectual function was unimpaired.

This was her third pregnancy; her two previous children, both healthy, had been delivered by emergency Caesarean section under general anaesthesia. She now strongly requested a regional anaesthetic technique, wanting to participate in childbirth as much as possible.

Inspection of the back revealed a big scar in the lumbosacral region, as well as kyphoscoliosis and obesity (Fig. 1). On palpation, the scarred region felt like a bony plate.

Cephalad to this, spinous processes were discernible. X ray pictures were not available at the time, but were neither considered to be decisive in the choice of anaesthetic technique, nor of much use in guiding lumbar puncture. Epidural anaesthesia was considered impossible and even if the space could have been identified would probably have resulted in incomplete blockade. It was thought that the puncture site and dose of local anaesthetic was unpredictable so it was decided to introduce a catheter in the subarachnoid space. The possibility of postdural puncture headache and the slight risk of nerve root or spinal cord trauma were explained to the patient who nevertheless willingly agreed.

On arrival in the operating theatre, 1 litre of compound sodium lactate solution was infused intravenously. Monitoring comprised the ECG and automatic noninvasive arterial blood pressure measurement. The patient was positioned sitting up and after local infiltration with lignocaine 1%, several attempts at dural puncture were made at different spinal levels. A bony resistance was encountered each time. The subarachnoid space was identified after 40 minutes of trial with an 18-gauge Tuohy needle, probably at the T₇₋₈ interspace. The needle bevel at that moment was oriented perpendicular to the midline and a 20-gauge catheter was advanced 3 cm without difficulty. The patient endured all this remarkably well.

One ml of hyperbaric bupivacaine 0.5% in glucose 8% was administered intrathecally. The patient was put in a semi-upright position to encourage caudal spread. Within 2 minutes sensory block, as determined by pinprick, extended

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Accepted 19 March 1990.



Fig. 1. The back of the patient, showing the lumbosacral scar and kyphoskoliosis.

from T₂ to T₁₂. Concomitantly, systolic arterial blood pressure decreased from 140 to 95 mmHg but responded quickly to ephedrine 2.5 mg intravenously. Sensory block extended caudally to L₁ after 15 minutes. The existing hypoaesthesia prevented proper testing in the sacral and lower lumbar segments. Surgery was started 15 minutes after the injection of the local anaesthetic. A healthy boy was delivered 10 minutes later with Apgar scores at 1 and 5 minutes of 9 and 10, respectively. The mother was allowed to hold the baby while the wound was closed.

Sensory block was receding 40 minutes after first injection of local anaesthetic and the patient experienced discomfort and pain in the lower abdomen. A top-up dose of 0.4 ml hyperbaric bupivacaine 0.5% was effective. The arterial blood pressure decreased once more to just below 100 mmHg and again was restored by ephedrine. The spinal catheter was removed in the recovery room. Recovery was uneventful, with no evidence of postdural puncture headache. She was discharged from hospital on the 10th postoperative day.

Discussion

Pregnancy in patients with spina bifida may become less uncommon, because as a result of intensive treatment they now reach adulthood.^{3,4} The number of reports of pregnancy in these patients has grown since the 1970s.³⁻⁸ Vaginal delivery is possible in selected patients⁹ but Caesarean section is usually preferred.⁷ We found two reports about regional anaesthesia in spina bifida patients: one case of epidural anaesthesia for vaginal delivery¹ and one of spinal anaesthesia for Caesarean section.⁹ This may indicate that in most cases general anaesthesia is used.

Epidural anaesthesia will be difficult and may result in unsatisfactory block or other complications in patients with a severely distorted spine.^{1,2} However, vertebral column abnormalities in themselves do not preclude spinal anaesthesia but may require puncture at a site other than usually used.¹⁰ The lack of versatility of spinal anaesthesia as compared to epidural¹¹ can be overcome by introducing a catheter in the subarachnoid space. This old technique is regaining interest.¹² The surprisingly low incidence of postdural puncture headache after the use of a spinal catheter in older patients¹³⁻¹⁵ has, as yet, not been confirmed in parturients. To minimise cerebrospinal fluid leakage and the chance of headache, the needle bevel should be parallel to the longitudinal dural fibres during puncture.¹⁶ The risks of introducing a catheter subdurally in the direct vicinity of the spinal cord are known.

In our patient, with hindsight a paramedian approach might have led to successful puncture at a lower spinal level. Moreover, the bevel should have been oriented parallel to the midline. For all that, there was no post-anaesthetic morbidity and she was very pleased with the technique.

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CASE REPORT

Severe hypotension due to intramyometrial injection of prostaglandin E₂

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Summary

Treatment of postpartum haemorrhage because of uterine atony with intramyometrial prostaglandin E₂ 1 mg, in an anaesthetised patient resulted in life-threatening hypotension. Anaesthetists should be aware of the potential cardiovascular side effects of prostaglandins.

Key words

Hormones; prostaglandins, PGE₂, PGF_{2α}
Haemorrhage.

Prostaglandins (PG) are well known to cause a decrease in arterial pressure. Such a case is reported here where severe hypotension followed injection of PGE₂ directly into the myometrium.

Case history

The patient was a 35-year-old primigravida with an uncomplicated twin pregnancy. Labour was induced by artificial rupture of the membranes and an intravenous infusion of syntocinon at 38 weeks' gestation. Seven hours later, the first twin was delivered by midcavity forceps and the second by breech extraction under epidural anaesthesia, which had been used for pain relief throughout labour. The estimated blood loss at delivery was 500 ml.

Two hours after delivery, she had a postpartum haemorrhage of about 1 litre because of uterine atony that failed to respond to intravenous syntocinon and ergometrine. She was given compound sodium lactate 1 litre intravenously and polygeline (Haemaccel) 500 ml and then underwent uterine exploration under general anaesthesia. Her arterial blood pressure before induction was 135/85 mmHg and heart rate 85 beats/minute. Anaesthesia was induced with thiopentone 300 mg after pre-oxygenation of her lungs. This was followed by suxamethonium 100 mg and the trachea was intubated. Anaesthesia was maintained with 1% enflurane and 65% nitrous oxide in oxygen supplemented by fentanyl 50 µg, and neuromuscular blockade with atracurium 20 mg.

Arterial blood pressure and heart rate remained stable at 120/80 mmHg and 70 beats/minute respectively during the

first 20 minutes of surgery. A further 500 ml of intravenous fluid were administered. The diagnosis of uterine atony was confirmed, and in an attempt to stimulate uterine contraction the obstetrician injected prostaglandin E₂ (Prostin E₂, Upjohn Ltd) 1 mg diluted in 10 ml 0.9% saline blindly through the abdominal wall into the myometrium. The patient's arterial blood pressure decreased 3 minutes later to 65/35 mmHg and then to an unrecordable level over the next 2 minutes. Her heart rate increased to 85 beats/minute. The carotid artery pulse was palpable, but not the peripheral pulses. The ECG monitor showed sinus rhythm with depression of the previously normal S-T segments. The patient was flushed and venodilated, but had no urticaria. Both lungs were being ventilated satisfactorily. Airway pressure was unchanged and there was no wheezing.

Enflurane was discontinued and the lungs ventilated with 100% oxygen. Plasma protein solution 1.6 litres were infused rapidly. Adrenaline 1 in 10 000 was given intravenously in 1-ml increments to a total of 5 ml; hydrocortisone 100 mg was also given. Arterial pressure was 70/30 mmHg and heart rate 120 beats/minute after 5 minutes. Fifteen minutes later these were 120/60 mmHg and 90 beats/minute respectively. The ECG had returned to normal. Nitrous oxide and enflurane were reintroduced. The results of a pre-operative full blood count and coagulation screen now became available. Haemoglobin was 6.7 g/dlitre, platelet count 134×10^9 /litre, and coagulation was normal. A transfusion of 5 units of packed red blood cells was started. There was little further bleeding from the uterus that was now well contracted. A uterine pack was inserted. The remainder of the procedure was uneventful, and after

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Accepted 19 March 1990

reversal of residual neuromuscular blockade the tracheal tube was removed when spontaneous ventilation had returned and the patient was awake.

A repeat coagulation screen after the operation showed prothrombin time 19 seconds (control 17 seconds), activated partial thromboplastin time 43 seconds (control 32 seconds), thrombin clotting time 11 seconds (control 11 seconds) and 10 units of cryoprecipitate were transfused. Unfortunately, uterine atony returned and 3 hours later the patient underwent a hysterectomy because of further bleeding. She ultimately made a good recovery.

Discussion

Prostaglandins are widely used in obstetrics for their stimulant action on the uterus. Prostaglandin E₂ (PGE₂) and prostaglandin F_{2α} (PGF_{2α}) may be given by the oral, intravenous and intra-uterine routes for induction of labour or therapeutic abortion.

PGE₂ and PGF_{2α} have different effects on the cardiovascular system.^{1,2} PGE₂ causes vasodilatation and reduces systemic vascular resistance. The systemic arterial blood pressure decreases but cardiac output is raised by increases in both heart rate and stroke volume; pulmonary vascular resistance is reduced. PGF_{2α} increases heart rate, systemic arterial blood pressure and cardiac output, but systemic vascular resistance is unchanged and pulmonary vascular resistance is increased.

Intramyometrial injection of PGF_{2α} has been shown to be an effective treatment for postpartum haemorrhage in patients with uterine atony unresponsive to syntocinon, ergometrine and uterine massage.³⁻⁵ PGF_{2α} 1 mg is usually given, but 0.25 mg has also been shown to be satisfactory.³ Intravenous infusion and intramuscular injection are not completely effective,³ while intramuscular⁶ or intramyometrial⁷ injection of 15-methyl PGF_{2α}, a more potent uterotonic and longer acting analogue of PGF_{2α}, are reported to be efficacious. It is suggested that these drugs are used early to obtain maximum effect and several doses may be needed. Side effects reported in these studies include flushing, headache, abdominal pain and hypertension. Arterial desaturation from acute increases in intrapulmonary shunting was described after intramuscular and (or) intramyometrial injection of therapeutic doses of 15-methyl PGF_{2α}.⁸ In one report,⁹ an overdose of PGF_{2α} (40 mg) by intramyometrial injection caused hypotension and pulmonary oedema.

Little is written about the use of PGE₂ to treat postpartum haemorrhage, but its successful use by vaginal suppository was reported in one patient;¹⁰ moderate diastolic hypotension occurred. Intravenous infusion of PGE₂ 10–20 µg/minute was also used successfully in a single case;¹¹ it caused an increase in heart rate but not hypotension. However, the intramyometrial administration of a large dose of PGE₂ (5 mg) to one patient caused severe hypotension.¹²

Intramyometrial injection of PGE₂ 1 mg for uterine atony resulted in profound hypotension in our patient, which was probably the result of vasodilatation after rapid systemic absorption of the drug. It is unlikely that hypovolaemia was a major contributing factor because arterial blood pressure and heart rate had been stable during induction and for the first 20 minutes of anaesthesia. Hypotension was treated effectively with a rapid infusion of plasma and intravenous adrenaline but a pure vasoconstrictor such as methoxamine might have been a more appropriate drug.

Anaesthetists should be aware of the problems that may be encountered with the use of prostaglandins in obstetric practice and of the differing physiological actions of PGE₂ and PGF_{2α}.

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CASE REPORT

Laryngeal oedema from a neck haematoma

A complication of internal jugular vein cannulation

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Summary

Laryngeal oedema occurred after formation of a neck haematoma after attempted internal jugular vein cannulation. This resulted in complete respiratory obstruction and respiratory arrest and it was impossible to ventilate her lungs manually or intubate her trachea. Oxygenation of the patient was only possible using transtracheal ventilation.

Key words

Complications; laryngeal oedema, haematoma.

Anatomy; internal jugular vein.

Respiratory obstruction during pregnancy is an uncommon condition usually only associated with extubation of the trachea.^{1,2} We recently encountered a patient in whom the development of laryngeal oedema from a neck haematoma led to respiratory arrest.

Case history

A 28-year-old woman presented at 35 weeks' gestation in her second pregnancy with a history of severe abdominal pain of one hour's duration. She was peripherally shut down and felt cold on examination. Her heart rate was 110 beats/minute, and arterial blood pressure 100/60 mmHg. She had no obvious peripheral or facial oedema. Her uterus was wooden hard and tender to palpation. There was no vaginal blood loss and no fetal heart could be heard. A diagnosis of concealed abruption and hypovolaemic shock was made. Urinalysis showed microscopic haematuria and proteinuria (+++). Initial blood results showed normal electrolytes, urea 4.2 mmol/litre, albumin 32 g/litre, urate 0.32 mmol/litre, haemoglobin 112 g/litre, platelets 242×10^9 /litre. Her clotting screen was normal: thrombin time 19 seconds, prothrombin time 15 seconds, kaolin time 50 seconds. Fibrinogen degradation products were less than 40 µg/litre.

Two intravenous infusions were started in view of her hypovolaemic state. A central venous catheter was inserted to guide fluid replacement. It proved impossible to enter a basilic vein and therefore the right internal jugular route was chosen, but unfortunately the right carotid artery was punctured. The attempt was made using the Seldinger

technique (Vygon Leadercath). Bleeding was stopped by applying pressure to the site for 10 minutes. A second attempt at right internal jugular catheterisation was successful. Her central venous pressure (CVP) was 0 cm H₂O. She received one litre of compound sodium lactate solution and one litre of polygeline before a blood transfusion was started.

Labour was induced by rupturing the mother's membranes and the liquor was noted to be heavily blood stained. Her analgesia during labour consisted of a bolus dose of diamorphine 5 mg intravenously followed by an infusion at a rate of 2 mg/hour (total 13 mg diamorphine given). Droperidol 2.5 mg was also given intravenously. She delivered a dead 2.63 kg male infant after one hour. There was a measured blood loss of 3000 ml at delivery and onset of frank haematuria. Further haematological investigation revealed haemoglobin 87 g/litre, platelets 88×10^9 /litre and all clotting times greater than 2 minutes. Fibrin degradation products were greater than 560 µg/litre and confirmed the diagnosis of disseminated intravascular coagulation. Further blood and coagulation factor replacement (as fresh frozen plasma) were given. She received a total of nine units of blood and four units of fresh frozen plasma.

She complained of difficulty in breathing during her labour. Auscultation of her chest confirmed adequate air entry, and pulse oximetry showed 99% saturation. Her respiratory rate was 18/minute; she had a clear sensorium, was anxious, but without any stridor. Thirty minutes after delivery she again complained of difficulty with breathing. There was blood oozing from the puncture site in her neck,

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Accepted 1 March 1990.

and her face and neck were swollen. She rapidly lost consciousness, became cyanosed and had ventilatory arrest. It proved impossible to ventilate her lungs despite an oral airway and triple manoeuvre.^{3,4} An attempt at tracheal intubation was made, because of a decline in oxygen saturation to 72%, but proved impossible. The vocal cords could not be visualised and there was gross oedema of the tongue, epiglottis and pharyngolaryngeal tissues. The patient regurgitated at this point despite 'cricoid pressure' (the cricoid cartilage was impalpable). A 14-gauge Argyll Medicut cannula was inserted into her trachea two finger-breadths above the sternal notch, in view of our inability to ventilate her lungs or intubate her trachea and the inability to discern anatomical landmarks in her neck. Aspiration through a water-filled syringe confirmed correct placement. The cannula was connected via a 3.5-mm Portex tracheal connector to a Bain system and the emergency bypass oxygen was turned on (35 litres/minute). Her saturation rapidly returned to 100%. The patient never became pulseless throughout the period of apnoea.

A further attempt at tracheal intubation was made 10 minutes later using a gum elastic bougie; this was successful but it was difficult to pass the 8.0-mm Portex tracheal tube into the trachea. It was still impossible to view the vocal cords although a swollen epiglottis could be seen. Tube position was confirmed by capnography. Aspiration of her trachea revealed no acidic or solid material. She was transferred to the intensive care unit at the main hospital in view of the disseminated intravascular coagulation and respiratory difficulties. She was sedated with a propofol infusion and her lungs were ventilated for 18 hours. She maintained excellent arterial blood gases with an inspired oxygen of 30% and her chest X ray showed no evidence of aspiration. Her coagulopathy was corrected. The next morning she was awake and breathing spontaneously via a T-piece and exhibited no neurological sequelae to her period of hypoxaemia.

A trial of extubation was decided upon. She was anaesthetised with 100% oxygen and halothane, and when deeply anaesthetised a gum elastic bougie was threaded down the tracheal tube and left in place. Laryngoscopy was performed and showed a swollen oedematous tongue, oropharyngeal tissues and vocal cords. Removal of the tracheal tube, leaving the bougie in the trachea, resulted in total respiratory obstruction. Re-intubation was performed with a 7.0-mm tracheal tube over the gum elastic bougie. There was a slight leak around the tube with this in the absence of cuff inflation, whereas with the previous 8.0-mm tube there had been no leak. The next day, under similar circumstances, a further trial of extubation was performed and on this occasion the airway was adequately maintained. The patient reported no further respiratory difficulties during her hospital stay or at her 6-week postnatal visit.

Discussion

Laryngeal oedema is an unusual complication during pregnancy. Known associations are pre-eclampsia,⁵⁻⁸ strenuous labour,⁷ raised venous pressure,⁷ overhydration,⁹ upper respiratory tract infection,¹⁰ excessive weight gain,¹¹ postextubation,¹² drug reaction,¹³ and Cl esterase inhibitor

defect.¹⁴ Other potential causes could be a blood transfusion reaction¹⁵ or a neck haematoma.¹⁶

This patient was not pre-eclamptic, her weight gain was as expected for a 35-week pregnancy, her labour was short and no evidence of respiratory tract infection was present. She had no family history of angioedema, had never developed bronchospasm, hypotension, or urticaria, and had no evidence of histamine release. Puncture of her carotid artery and the coagulopathy she developed certainly led to a neck haematoma, which at the time we did not think could precipitate this respiratory difficulty and certainly not without stridorous breathing. We presume that blood must have tracked out of the carotid sheath and into the paratracheal tissues to cause venous obstruction in her neck. This resulted in gross laryngeal oedema. Of the 17 cases of laryngeal oedema that relate to pregnancy reported in the literature only three exhibited signs of stridor.^{1,2,10} It therefore appears that stridor is an uncommon finding even when the laryngeal oedema ultimately results in a total respiratory obstruction.²

We believed that central vein cannulation was appropriate in this patient because of the substantial blood loss involved. It proved impossible to cannulate the basilic vein because of her peripheral shutdown, even so the success rate of correct placement of basilic vein catheters into the superior vena cava is only 70%.¹⁷ Internal jugular vein cannulation has the highest success rate; the incidence of carotid artery puncture is 2%,¹⁷ although this seldom causes problems. It is, however, inadvisable in patients with coagulopathies.¹⁸ Carotid artery puncture has never been documented to cause laryngeal oedema of such a degree as to result in a respiratory arrest.

The inability to oxygenate this patient was only overcome via the technique of transtracheal ventilation.^{4,19} A Portex minitracheostomy set was available, but the inability to discern anatomical landmarks in the neck made its use inadvisable.²⁰ Transtracheal ventilation allowed adequate oxygenation but could not guarantee carbon dioxide clearance. It did allow the attending anaesthetists time to consider the management options available. In the absence of a fiberoptic bronchoscope if we had failed to intubate her trachea then tracheostomy would have been the only option left.

The patient regurgitated but there was no evidence that she aspirated, probably because of the complete respiratory obstruction. This was fortunate because she had not received antacid or H₂ blocker prophylaxis. This was remiss on our part and emphasises the need for their administration in at-risk patients.

This is an unusual case of laryngeal oedema that led to total respiratory obstruction, without signs of stridor. Its aetiology was even more uncommon because it was the result of a neck haematoma. This risk of laryngeal oedema must be borne in mind if the carotid artery is punctured in a patient who subsequently develops a coagulopathy. A complaint of difficulty in breathing by the patient should then be regarded as a sign for early intubation before complete respiratory obstruction ensues. In these circumstances of total respiratory obstruction only transtracheal ventilation via a catheter directly into the trachea will successfully oxygenate the patient. We consider that some means of performing this should be available in areas

where anaesthetics are given, preferably with a Saunders Venturi injector to attach to it.¹⁹

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CASE REPORT

Adverse haemodynamic effects of high-dose aprotinin in a paediatric cardiac surgical patient

H. BÖHRER, A. BACH, F. FLEISCHER AND J. LANG

Summary

High-dose aprotinin for reduction of intra- and postoperative blood loss was associated with profound hypotension and flushing in a 3.5-year-old child who underwent cardiac surgery. Treatment with noradrenaline and intravenous fluid was required. Cardiovascular stability was restored after 10 minutes.

Key words

*Complications; hypotension.
Blood; coagulation.*

Several techniques including haemodilution and autotransfusion have been used to reduce blood loss and the need for transfusion during and after open-heart surgery. Pharmacological intervention with desmopressin^{1,2} or dipyridamole³ has been suggested to reduce early postoperative blood loss and transfusion requirements. Recently, several groups have shown that the kallikrein inhibitor aprotinin improves haemostasis after cardiopulmonary bypass.⁴⁻⁹

We have used high-dose aprotinin routinely during cardiac surgical procedures in adults. We have extended this regimen in a pilot study to paediatric cardiac patients. We report the case of a child who experienced a sudden unexpected decrease in arterial pressure shortly after high-dose aprotinin.

Case history

A 3.5-year-old, 12-kg boy was scheduled for pulmonary artery debanding and patch closure of a ventricular septal defect. As an infant, he had undergone ligation of a patent ductus arteriosus, pulmonary artery banding, and patch grafting of an aortic coarctation.

The patient received ketamine 35 mg and atropine 0.15 mg intramuscularly as premedication. Ten minutes later, an intravenous cannula was inserted into a vein on the dorsum of the right hand. Anaesthesia was then induced with fentanyl 0.25 mg, diazepam 2.5 mg, and pancuronium 1.5 mg intravenously. The trachea was intubated, and ventilation was controlled with a Servo 900 C ventilator (Siemens-Elema, Sweden) set at an inspiration/expiration ratio of 1:2

and a rate of 20 cycles per minute starting with an F_{IO_2} of 0.5 (F_{IN_2} 0.5). Arterial oxygen saturation (SpO_2) measured by a Nellcor N-100 pulse oximeter remained stable between 97 and 99%. The heart rate and arterial pressure remained unchanged at 130 beats/minute and 115/60 mmHg, respectively, throughout the induction period. A double-lumen central venous catheter was inserted via the right internal jugular vein; the central venous pressure was 13 mmHg. In addition, an arterial line was placed in the right radial artery.

Forty-five minutes after induction, 625000 KIU of aprotinin (Trasylol, Bayer) were infused intravenously over 5 minutes. No other medications were given during this period. A sudden decrease in arterial pressure from 115/60 to 50/30 mmHg was noted approximately one minute after the end of the aprotinin infusion, followed by generalised marked flushing of the skin. SpO_2 decreased from 98 to 90% despite an increase in F_{IO_2} to 1.0. The heart rate remained at 125 beats/minute. Noradrenaline 30 μ g was administered intravenously and Ringer's solution 300 ml was infused over the next 5 minutes. The arterial pressure increased to 90/60 mmHg. In addition, steroids were given intravenously. Another bolus of noradrenaline (10 μ g) was required to keep the systolic arterial pressure at 90 mmHg. Sodium bicarbonate 10 mmol was administered because of metabolic acidosis. Ten minutes after the event, the cardiovascular system became stable, and the rash began to disappear. Arterial blood gases reached baseline values, and the acid-base status was in balance. It was decided to begin with the scheduled operation, which was concluded

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Accepted 28 February 1990.

successfully. The patient's postoperative course was unremarkable, and his trachea was extubated on the first postoperative day.

Discussion

Untoward reactions occur frequently in the peri-operative period because of the multitude of drugs administered.¹⁰ The majority of these reactions are transient and not associated with significant morbidity or mortality. However, hypotension and cardiovascular collapse in association with generalised flushing may have serious consequences if no adequate therapeutic measures are taken. The haemodynamic reaction and flushing in our patient started shortly after the end of the infusion of aprotinin. No other medications had been given in the previous 45 minutes, which suggested that the hypotension was related directly to high-dose aprotinin administration. It is very unlikely that unrecognised hypovolaemia or acute myocardial depression contributed to the hypotension.

Aprotinin is a naturally occurring inhibitor of proteolytic enzymes. It is a polypeptide with a molecular weight of 6512 daltons which is isolated from bovine lung and other tissues.¹¹ The activity and concentration of the commercial preparation is given in KIU (kallikrein inactivator units). The mechanism by which aprotinin acts on haemostasis during and after cardiopulmonary bypass is not completely known. It is thought to preserve platelet function and thus reduce peri-operative blood loss and transfusion requirements.^{4,9} Aprotinin is usually given as a bolus approximately 20 minutes before sternotomy, followed by a continuous infusion. An additional bolus is added to the priming volume of the extracorporeal circuit.⁸

The nature of the allergic response to aprotinin seen in our patient remains unclear. As a foreign protein, aprotinin may cause mediator release from mast cells and basophils. In an anaphylactoid reaction, histamine liberation is related directly to the dose and the speed of administration of the drug.¹² The rapid and high-dose infusion of aprotinin in our patient may have caused degranulation of mast cells and basophils. The resulting clinical adverse effects consisted of transient flushing and hypotension, which required treatment with noradrenaline, and fluid replacement.

We have described a case in which high-dose aprotinin, administered to improve haemostasis following cardiopulmonary bypass, was associated with hypotension in a paediatric cardiac surgical patient. Thus, the decision to use aprotinin in children should be weighed against its

disadvantages, and special attention should be given to potential adverse reactions. Further studies are required to determine the mechanism of this adverse haemodynamic effect of aprotinin.

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Mechanical ventilation during low-flow anaesthesia

Experience with an alternative to the bag-in-bottle

L. BERNTMAN, H. H. LUTTROPP AND O. WERNER

Summary

Clinical experience with low-flow anaesthesia during controlled ventilation of the lungs is described. The anaesthesia circle is separated by a corrugated hose that serves as a large deadspace. This open connexion has no bellows or overflow valve and therefore the risk of mechanical dysfunction is small. No mixing of circle and ventilator gas occurs during normal operation. Major decreases in the oxygen concentration in the system are unlikely even if the fresh gas flow is interrupted or significant leaks from the circle occur because 100% oxygen is delivered by the ventilator. A hose volume larger than 1650 ml prevented gas mixing at tidal volumes of 380–1170 ml. There was no system-related mishap in over 600 patients, who comprised about 40% of the neurosurgical patients anaesthetised during that period. The cost of isoflurane was reduced to about 33% of that incurred during previous periods.

Key words

*Anaesthetic techniques; closed system.
Ventilation; artificial.*

Low-flow anaesthesia (LFA) has recently regained popularity. One reason for this is the reduced consumption of isoflurane, and another that LFA requires gastight systems, which reduce gas pollution in the operating room. Finally, LFA helps to reduce emissions of halogenated hydrocarbons and nitrous oxide into the atmosphere.

Mechanical control of ventilation with LFA is usually achieved by a bag-in-bottle arrangement, i.e. the ventilator gas is separated from the patient circle by bellows inside an airtight container. The function of this device is simple, but critically dependent on proper function of the bellows and the circle's overflow valve.

We used an old method^{1,2} as an alternative whereby the ventilator is connected to the anaesthesia circle via a large deadspace that prevents ventilator gas from entering the circle. This is a safe and simple method that combines the facilities of a powerful ventilator with the advantages of the circle system. This paper presents the principles of operation, bench experiments, trials in pigs, evaluation in clinical practice, and economy.

Materials and methods

Principle of operation

The ventilator (Servo 900 C, Siemens-Eléma, Solna, Sweden), delivers oxygen into a large deadspace that consists of a corrugated polyethylene (Hytrel) hose with 2.2

litres internal volume. The other end of the hose is connected to a conventional anaesthesia circle with a CO₂ absorber (Monosorb, Siemens-Eléma, Solna, Sweden), one-way valves and fresh gas inlet (Fig. 1). The ventilator pushes a tidal volume of oxygen into the deadspace and the same volume of anaesthetic gas mixture is expelled from the other end into the circle. The gas column in the hose oscillates back during expiration: a tidal volume of exhaled gas is collected in the circle end of the deadspace, while one tidal volume of oxygen, mixed with some excess gas from the circle leaves via the ventilator. The pressure generated by the ventilator is transmitted unchanged to the circle since the hose is wide (3.3 cm internal diameter). In essence, the setting on the ventilator (minute volume, frequency etc.) will determine the pattern of ventilation, while the gas composition in the circle is determined by the vaporizer and flowmeter settings connected to the circle's fresh gas inlet. The different ventilatory modes and other functions of the ventilator can be used without restriction. Ventilation volumes, frequency etc. are monitored on the display of the ventilator. However, there is some discrepancy between the inspiratory and expiratory volumes as measured in the ventilator and volumes inhaled and exhaled by the patient. Thus, the fresh gas flow that enters the circle during the inspiratory phase of the ventilator, i.e. when the expiratory valve is closed, will increase the inhaled volume, while fresh gas entering the circle during expiration will increase the flow through the expiratory

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Accepted 22 February 1990.

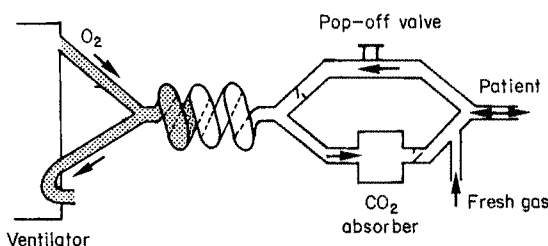


Fig. 1. The low-flow system. The pop-off valve is functionally closed (35 cm H₂O) during mechanical ventilation.

flowmeter of the ventilator, although no corresponding volume is exhaled. With the standard setting of the respiratory cycle, i.e. inspiration time 25% and pause time 10%, 35% of the fresh gas flow (FGF) will be added to the inspired minute ventilation and the rest will bypass the patient but be monitored by the ventilator.

Bench tests

Experiments were undertaken to evaluate the deadspace volume necessary to prevent mixing of ventilator gas with that of the circle. The fresh gas flow was set to one litre/minute of 50:50 N₂O/O₂. The circle was connected to a lung model (a 4-litre anaesthetic bag). Oxygen concentration in the circle was measured with an oxygen analyser (Hudson Ventronics 5590, Temecula, California, USA) placed in the expiratory limbs. The ventilator delivered 10 litres/minute of O₂. Tidal volume (*V_T*) was varied from 380 to 1170 ml by changing the ventilator frequency. The hose volume (*V_D*) was stepwise increased from 550 to 2200 ml and the reading on the oxygen analyser was noted.

The compressible volumes were measured at different *V_D* by obstructing the Y-piece, then noting the expiratory volume (*V_E*) at a plateau pressure of 2.0 kPa.

Tests in pigs

The tests compared the low-flow system's isoflurane requirements with those of a non-rebreathing system. Four pigs (18.5–25 kg) were studied. Anaesthesia was induced with azaperon (Stresnil) and etomidate chloride (Hypnodil) and the tracheas of the pigs were intubated. The minute ventilation (4–6 litres/minute at a respiratory rate of 10 minute) was adjusted to give an end-tidal carbon dioxide concentration of approximately 4.5%, as measured at the tracheal tube on a Normocap analyser (Datex, Finland). Gas sampled by the analyser was returned to the circle. Fresh gas flow during LFA was one litre/minute of 50:50 N₂O/O₂ with isoflurane. End-tidal isoflurane concentration (Servo Gas Monitor, Siemens, Sweden) was kept at 1.5%. Isoflurane anaesthesia was maintained for 2 hours. The ventilator in the non-rebrea-

thing system supplied 50:50 N₂O/O₂ with isoflurane directly to the pig. Isoflurane concentration and duration of anaesthesia were the same as above. Isoflurane consumption was assessed by weighing the vaporizer on a precision balance (Galaxy 1200-SO, Ohaus, USA). The experiments were done in random order and separated by a one-hour wash-out phase.

Clinical use of the system

The system was used in routine anaesthesia in over 600 adult patients scheduled for neurosurgical procedures that lasted up to 14 hours. Patients were unselected except that those with known severe lung dysfunction were excluded. Anaesthesia was induced by thiopentone and maintained by N₂O, fentanyl and 0.5–1% isoflurane. The circle was connected to the ventilator via the corrugated hose after tracheal intubation. The pop-off valve in the circle was set at 3.5 kPa; this was closed functionally since airway pressures normally do not reach this level. The fresh gas flow was 1.5 litres/minute of oxygen and 3 litres/minute of nitrous oxide with isoflurane as needed during the initial 20–30 minutes that were spent in the induction room. The higher flow was used for denitrogenation and to allow for the patients' initial high uptake of nitrous oxide. The flowmeters were set to 0.5 litres/minute each of O₂ and N₂O for the duration of anaesthesia after transfer into the operating theatre.

Respiratory monitoring consisted of expired minute volume and airway pressures as displayed by the ventilator, and O₂ and CO₂ concentrations sampled from the tracheal tube into a Normocap (Datex)³ which withdrew 150 ml minute from the circle. The sampled gas was diverted to the scavenging system, after passing the analyser, instead of being delivered back to the circle. Isoflurane concentration was measured (Servo Gas Monitor, Simens, Sweden) at the tracheal tube in most patients and the sampled gas returned to the circle. Anaesthesia gases were discontinued after surgery, and oxygen flow via the fresh gas inlet was increased to 6 litres/minute. The circle was separated from the corrugated hose when the patient was ready for spontaneous breathing and instead reconnected to an anaesthesia bag.

Results

Bench-tests

Figure 2 shows the oxygen concentration in the circle at different tidal (*V_T*) and hose (*V_D*) volumes. Oxygen concentration was unaffected by changes in *V_T* when *V_D* was 1650 or 2200 ml. When *V_D* was reduced to 1100 ml significant mixing occurred at tidal volumes of or above 765 ml. The results at a *V_D* of 550 ml suggested significant mixing with oxygen from the ventilator at all tidal volumes tested.

The calculated compressible volumes of the system, at different values of *V_D* are given in Table 1.

Tests in pigs

Isoflurane consumption during 2 hours of anaesthesia was 0.4 (SD 0.1) (g/kg)/hour when a low-flow technique was used. In the non-rebreathing system the isoflurane consumption was 1.9 (SD 0.1) (g/kg)/hour.

Table 1.

<i>V_D</i> (ml)	<i>V_C</i> (ml/kPa)
550	0.22
1100	0.26
1650	0.29
2200	0.33

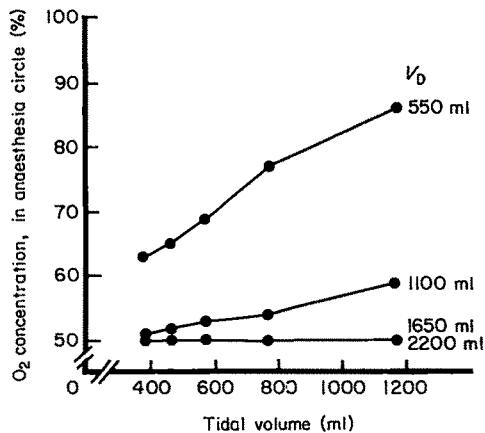


Fig. 2. Oxygen concentration (mean of duplicate experiments) in the circle at different tidal and hose volumes (V_b). Fresh gas flow to the circle was one litre/minute of 50% oxygen in nitrous oxide. The ventilator minute volume was 10 litres. Mixing of ventilator oxygen with circle gas is indicated by an increase in the measured oxygen concentration.

Clinical experience

The low-flow system has been used since August 1987 without any system-related incidents. From September 1987 low-flow anaesthesia was used in 60% of elective, daytime neuroanaesthesia cases. Figure 3 shows the cost for isoflurane per each 6 month-period from 1985 to 1989. Approximately 62 000 SEK (about £5600) was spent on isoflurane during the first 6 months of 1987 while the corresponding sum for 1988 was 26 000 SEK (approximately £2300), in spite of a 15% increase in total anaesthesia time at the unit.

Discussion

Closed-system and low-flow anaesthesia have been used for many years, but many anaesthetists prefer non-rebreathing systems because of their greater simplicity. The development of in-circle vapour and O_2/CO_2 analysers with alarms have increased the patient safety during LFA and permit anaesthetists to attend to other duties during the procedure.

An advantage of the present system is that moving parts such as bellows and a special overflow valve are not needed. This minimises the risk of mechanical dysfunction. Cleaning the system is simple. A possible disadvantage is that the absence of bellows that move may make the system intuitively less easy to understand than a bag-in-bottle ventilator.

During steady-state anaesthesia expired minute ventilation (\dot{V}_E) at the ventilator will be: the inspired minute ventilation (\dot{V}_I) set at the ventilator + fresh gas flow—oxygen consumption—gas consumed by the gas analysers. If the patient consumes 250 ml O_2 per minute, the analyser samples 150 ml/minute and fresh gas flow is one litre/minute the measured \dot{V}_E will be 0.6 litres/minute larger than the set \dot{V}_I . The exact oxygen concentration in the circle will be a function of the composition and flow of fresh gas, the oxygen consumption and anaesthetic gas uptake^{4,5} and the amount of gas removed via gas analysers. We usually obtain an oxygen concentration of 41–43% in the circle at steady state when the flowmeters are set at 0.5

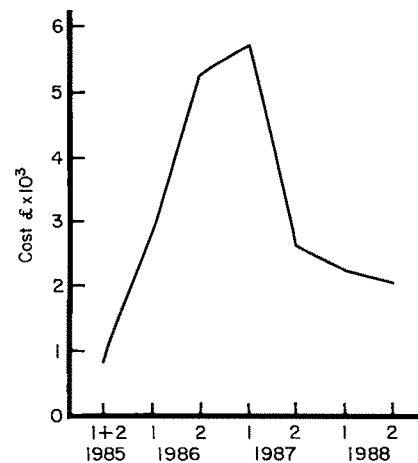


Fig. 3. Cost of isoflurane for the first (1) and second (2) 6-month period, 1986–88; the total expense is given for 1985.

litres/minute of N_2O and 0.5 litres/minute of O_2 . Some modifications were introduced after the initial evaluation of the system, for example the gas from the CO_2 monitor is now returned to the circle and the oxygen concentration in the circle is adjusted to about 35% by reducing the oxygen supply.

A hose volume (V_b) of 1650 ml prevented entry of ventilator oxygen into the circle, using a clinically relevant range of adult tidal volumes. We elected to use 2200 ml in order to increase the margins.

A low-flow system should be gas tight for proper function, but leaks may nevertheless occur e.g. around the cuff of the tracheal tube. The result of a leak in the present system varies with the leak volume (\dot{V}_L). If \dot{V}_L is smaller than the excess gas in the circle (in our case approximately 0.6 litres/minute, see above), measured \dot{V}_E will decrease slightly, but there will be no change in oxygen concentration in the circle. If \dot{V}_L is larger, O_2 from the ventilator will enter the circle and cause an increase in the oxygen concentration. A disconnection anywhere in the system will immediately be recognised as a decrease in measured \dot{V}_E .

The pressure in the system is controlled by both the setting of the pop-off valve and that of the high-pressure limit on the ventilator. We usually set the high-pressure limit below that of the pop-off valve, and it could be argued that we could as well close the pop-off valve entirely when switching from manual to mechanical ventilation (Fig. 1).

Interruption of fresh gas supply is unlikely to cause hypoxia, because under these conditions the oxygen and N_2O uptake by the patient will cause a net flow of oxygen from the ventilator via the hose towards the anaesthesia circle. Oxygen concentration will soon increase above the previous level (Fig. 4) after an initial decrease. However, interruption of only the oxygen while the nitrous oxide flow remains will cause hypoxia if this accident is not detected by the O_2 analyser.

The system has the vaporizer outside the circle. With this arrangement the inhaled vapour concentration may be considerably lower than that set on the vaporizer. The difference is dependent on FGF, ventilation and the patient's uptake of vapour. The concentration can and should be monitored by an analyser, sampling gas from the tracheal tube, to minimise the risk of intra-operative awareness. This is particularly important when an inhalant-only

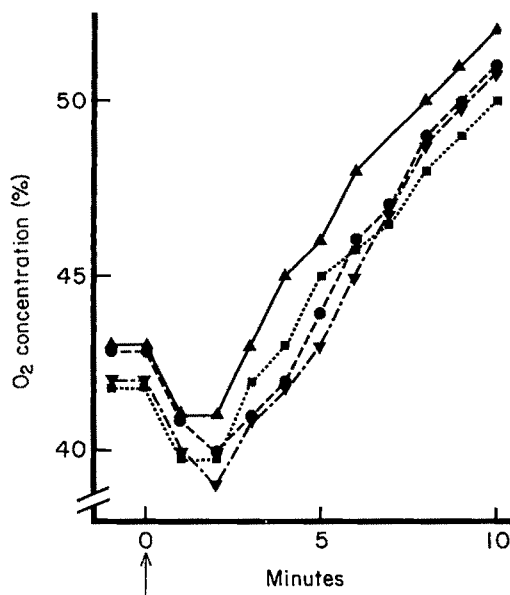


Fig. 4. Changes in O_2 concentration in the circle after turning off (at arrow) the fresh gas flow. Four trials in the same patient.

anaesthetic is given. Our patients all had a N_2O -fentanyl background and in most patients the isoflurane concentration was monitored.

The low-flow system was used in 60% of elective day-time neurosurgical operations, which comprise about 40% of the total case load. The cost of isoflurane in the neuro-anaesthesia service was reduced to 33% of that when only non-rebreathing systems were used, in spite of a 15% increase in anaesthesia time. The relatively larger reduction in cost than in the number of anesthetics with open systems is because of a preferential use of LFA with isoflurane for long operations.

A calculation of costs for maintenance anaesthesia (Fig. 5) (1% isoflurane, O_2 and N_2O) with open and closed systems is shown in Figure 5. LFA not only affects the cost of isoflurane, but also reduces the consumption of N_2O (cost 0.7 p/litre) and increases that of O_2 (cost 0.1 p/litre) which is used to drive the ventilator. In addition LFA requires a CO_2 absorber for £9. The two systems break even after 30 minutes, after which time the cost is about 15% of that of a non-rebreathing system. The calculations do not include costs for induction or reversal drugs and the comparison refers to maintenance cost after the break even

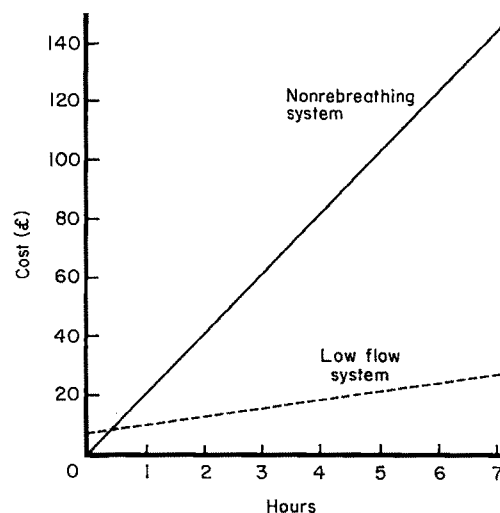


Fig. 5. Calculated cost of maintenance anaesthesia with 1% isoflurane in either the low-flow (0.5 litres O_2 /0.5 litres N_2O + 12 litres O_2 per minute, see text) or a non-rebreathing system (5 litres O_2 /7 litres N_2O per minute).

point. The isoflurane consumption (and cost) in our animal experiments in LFA was 22% of that using the open system. A retrospective study in humans⁶ compared total drug costs for relatively short procedures (about 2.5 hours) and showed that the cost for LFA was almost 50% of that with an open system. Gas costs become more and more important during longer procedures.

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The Woolley and Roe case

A reassessment

C. D. D. HUTTER

Summary

In 1953, two patients, Cecil Roe and Albert Woolley, sued their anaesthetist for alleged negligence because they had developed painful spastic paraparesis after spinal anaesthesia. The court found that phenol, which was used to sterilise the outside of the ampoules of local anaesthetic, had percolated the glass through invisible cracks, contaminating the solution, but that the anaesthetist could not have been aware of this risk. The case was important, despite the fact that judgement was in favour of the anaesthetist, because of the fears that it generated over the incidence of paralysis after spinal anaesthesia. The 'invisible crack' theory has been the subject of much scepticism. New information has been obtained, and the case re-examined objectively. The most probable source of contamination, which led to paralysis in the two patients, and in a third who received spinal anaesthesia on the same day, has been identified. A similar explanation may lie behind a number of other episodes of paralysis associated with spinal anaesthesia.

Key words

*Anaesthetic techniques; spinal.
History; Woolley and Roe case.*

The Woolley and Roe case made spinal anaesthesia unpopular, particularly in the UK, despite the fact that there were other reports of these neurological complications.^{1–5} Confidence has returned gradually since then, but the origin of these early paralyses has never been understood fully.^{6,7} This objective re-examination of the case has identified the most likely source of the problem in these two patients and may have revealed the most probable explanation for earlier episodes of paralysis that followed spinal anaesthesia.

Summary of the case^{8–12}

On Monday, 13 October 1947, two patients, Cecil Roe (aged 45 years) and Albert Woolley (aged 56 years), who were both in good health with no history of pre-existing neurological disease, each received spinal anaesthesia for removal of a displaced semilunar cartilage and repair of hydrocoele respectively. They were anaesthetised with 10 ml hypobaric cinchocaine 1:1500 in the same operating theatre by one anaesthetist.

Cecil Roe, the first patient in the morning, suffered severe pain in his head and back during the operation. He also experienced a burning sensation at the operative site as soon as the surgeon had started, which indicates that analgesia was only barely adequate. This is significant and will be discussed in detail later. The pain in his head and

back continued to be very severe until at least the evening (personal communication: family of Mr Cecil Roe). Albert Woolley was anaesthetised in the afternoon and experienced no discomfort whatever.

It was apparent by the next day that both patients had developed an acute myelopathy that involved the nerve roots of the cauda equina and the lower spinal cord where the concentration of spinal anaesthetic had been highest.¹³ Clinically, the myelopathy presented with flaccid paralysis of the legs, anaesthesia over the lower abdominal wall, legs and peri-anal region and incontinence of urine and faeces. The symptoms progressed to a painful spastic paraparesis after an initial improvement. There was never any meningism or evidence of meningitis. The course of the illness in the two patients remained very similar. This suggested a common aetiology, although Cecil Roe was always a little more badly affected than Albert Woolley. Later Cecil Roe underwent a laminectomy followed by intrathecal alcohol injections in an attempt to alleviate some of his symptoms.

A third patient also received hypobaric cinchocaine that morning, after Cecil Roe. He underwent surgery for intestinal obstruction and the spinal anaesthetic was administered by the same anaesthetist. The patient died 5 days after operation from the effects of his illness, but before this, he also began to show signs of spinal sequelae (personal communication Dr J.M. Graham and family of Mr Cecil

Roe). There were no further sequelae of this nature associated with spinal anaesthetics at the hospital after these three incidents.

Cecil Roe and Albert Woolley sued the Ministry of Health (as trustees of the hospital) and the anaesthetist for damages and in October 1953 the case came to court amidst considerable publicity.^{14,15} The case for the plaintiffs, which was supported by Professor Robert Macintosh, rested ultimately on the allegation that a solution of phenol, in which the ampoules had been immersed in order to maintain their sterility, had seeped through 'invisible cracks' in the glass and contaminated the cinchocaine. It was alleged that paralysis resulted from this contamination. This was the explanation accepted by the judge despite conflicting evidence from other witnesses.

The possibility of leakage into ampoules was well recognised at the date of the trial in 1953, although this was not so in 1947 when the anaesthetics were given. It was ruled that there had been no negligence, as a competent anaesthetist could not have been aware of this possibility at that time. Thus, the judgement went against the plaintiffs, who were not compensated. This decision was upheld by the Court of Appeal in 1954.

However, there was a strong body of medical opinion at the time which held that the explanation accepted by the judge did not represent the true facts. It was these doubts which helped spinal anaesthesia to fall into disrepute in this country.⁶

The details of the case

'Invisible cracks'

There is some evidence that if glass ampoules (without any visible signs of cracks or damage) are immersed in a liquid, then this liquid can percolate through the glass and contaminate the contents.^{4,16,17} But is this relevant? The ampoules used for all three patients were placed in phenol 48 hours before the operation.¹⁵ It seems exceptionally unlikely that percolation of an estimated 4 ml of phenol solution (an estimate presented to the judge, but based on no substantive evidence),⁸ could come about in so short a time. If such extensive percolation over this very short period could occur, then similar episodes would have been expected during the previous 9 months when it was hospital policy to immerse ampoules of cinchocaine in phenol.⁹ The fact that this is not so virtually excludes the possibility of percolation as the means by which these patients received their injuries. (This must not be confused with visible cracks. Ampoules were always carefully examined for these before use.)⁹

Phenol, its diluent, or any added dye can adhere to the wall of an ampoule,¹⁸ and may contaminate the contents when the ampoule is snapped open. This can be discounted by the same argument as a likely cause of these injuries.

Phenol

No one was aware, at the time of the trial, of the effects of a dilute solution of phenol in the subarachnoid space. However, phenol was known to be a chemical irritant, and presumably upon this basis, it was alleged that phenol had led to the extensive injuries suffered by the plaintiffs.⁹ The use of intrathecal phenol for treatment of chronic pain was

first reported one year after the trial ended,¹⁹ and later findings were documented extensively.²⁰⁻²² These show quite clearly that the effects of intrathecal phenol differ markedly from the symptoms displayed by the plaintiffs. The most striking instance is the demonstration that spasticity is a symptom which may be alleviated rather than caused by phenol.⁷

Consequently, it seems very unlikely that phenol was responsible for the neurological damage in the patients Woolley and Roe (or in the third patient). This view is supported by pathological evidence which is discussed below.

Cinchocaine

Both neurologists were adamant that phenol was not the cause of these injuries. However, they went on to blame either cinchocaine itself as the toxic agent, or 'spinal anaesthesia' in general.⁹ This opinion may have been valid for some of the older spinal anaesthetic drugs such as amylocaine (Stovaine) which were allegedly neurotoxic.²³ However, the use of cinchocaine continued until very recently, and it is recognised that the drug is not neurotoxic in normal clinical doses.²⁴ However, the neurologists had good grounds at that time to consider cinchocaine, or any other spinal anaesthetic as the harmful agent; this will become apparent when their evidence is examined.

Identity of the ampoules

The operating routine was to immerse ampoules in a jar of 1:20 phenol (12 ampoules to the jar)²⁵ in which the paper labels were allowed to soak off. They were then transferred, now completely without identification, to a jar of 1:40 phenol to await use. This raises the possibility that the wrong ampoules were placed in the first jar, but incorrect ampoules could not have been involved for four reasons.

Random placement of three wrong ampoules in the final jar in the first instance would have made it extremely unlikely for them to have been selected consecutively. If the postulated number is increased then consecutive selection on that Monday becomes more likely. However, there should then have been similar problems in the days that followed, since the same jar remained in use. It is not clear how many spinal anaesthetics were performed later that week before it was realised 3 days later^{11,25} that there had been a serious problem with the anaesthetics given on the Monday, but no further problems occurred.

Secondly, Cecil Roe, the first patient to receive spinal anaesthesia on that day, was much more seriously affected than Albert Woolley. This marked difference is not consistent with injection of an identical but toxic drug. Thirdly, the pharmacy and the operating theatre were searched for other 20-ml ampoules that could have been substituted. None was found. (Personal communication, Dr J.M. Graham.) There were, and still are, very few drugs dispensed in 20-ml ampoules. Finally, analgesia was just adequate in the first case, and quite satisfactory for the final two, which demonstrates that the ampoules must have contained a substance with local anaesthetic properties.

Consequently, it is almost certain that each patient received cinchocaine. Thus, paralysis must have been caused by some form of contaminant.

Source of contamination

The contaminant must have been introduced either before the ampoules were sealed, after the ampoules were sealed, or after the ampoules had been opened.

Other cases would have become apparent elsewhere, if the batch had been faulty, and the symptoms experienced by the patients would not have varied a great deal. Ciba, the manufacturers of the drug, in any case found no fault with the contents of other ampoules in the same batch.⁹ The contaminant could conceivably have been in a small number of ampoules before cinchocaine was added. However, the manufacturing process excludes this. In addition, it is extremely unlikely that the contaminated ampoules would appear later to be used in consecutive patients.

It is not possible to perforate the glass once the ampoule has left the manufacturer, without this becoming immediately apparent upon external examination. This theory can also be rejected.

Every other likelihood has been ruled out, so the contaminant must have been introduced after the ampoules had been opened. The syringes and needles are the only remaining source of contamination. The practice in 1947 was for needles and syringes to be washed only in water after use. Chemicals or detergents were never used. They were boiled for 20 minutes in tap water, in order to sterilise them before spinal anaesthesia, in a steriliser used solely for spinal anaesthetic instruments. The court was told that they were then removed, rinsed in sterile distilled water and placed on the spinal trolley. However, the practice in some hospitals at that time was to transfer the instruments directly to the spinal trolley without rinsing.

Thus the contaminant could have been present in the water which was used for washing the instruments. It cannot be proved that this was not the source of adulteration, but on balance the evidence must cause us to reject this suggestion. There is no particular reason why a chemical should have been placed in this water and contamination at this site does not fit in with the sequence of events which will be described below. Consequently contamination must have come from the steriliser.

The contaminant

The opinion of the neurologists at the trial was that these two cases, and other earlier ones, all formed a homogeneous group of patients who presented characteristic and recognisable features, and that the only known common factor to them all was the administration of a spinal anaesthetic drug.⁹ All attention was focused on the drug itself. However, there is also the possibility that the syringes and needles used to administer the drug contained a contaminant. The most likely source of contamination, and the factor common to them all, is the water in which they were boiled.

Water-boiling sterilisers all suffer the same problem of deposition of lime, or calcium carbonate, depending on the 'hardness' of the water,²⁶ and it is necessary for these deposits to be removed or 'descaled' by 'descalers' if they are to function properly. The most efficient method is with an acid descaler. No other maintenance procedure for sterilisers that requires the use of other chemicals has been identified, so the descaler was most probably the contami-

nating agent. The type supplied to the hospital at that time is not known; however, phosphoric and hydrochloric acids are examples of the acidic constituents of modern descalers. The General Hospital Nottingham (in a neighbouring district to the hospital in which these events occurred) used a preparation around the time of the incident that contained phosphoric acid 31% (3.2 molar).

There is no information about the effects of these acids on the central nervous system. The circumstances under which corrosive damage to nervous tissue may arise must be inferred from the details of mineral acid damage elsewhere in the body. The harmful effects of mineral acids depend firstly on their pH. A pH of 3.5 was cited in this context as capable of producing tissue irritation when inadvertently given subcutaneously.²⁷ Adhesive arachnoiditis was reported after the accidental intrathecal injection of 2-chloroprocaine. This drug is the most acidic of all the local anaesthetics (pH 3.3–3.4) and it was suggested that this may have been a significant aetiological factor.²⁸

Further information is available from examining the effects of acid injury to the eye. Destruction of nerve terminals with loss of sensation may be associated with ocular damage from an acid²⁹ and corneal lesions are produced by hydrochloric acid at or below a pH of 2.5.³⁰ This correlates with the general assumption that lung damage from inhalation of hydrochloric acid in stomach contents arises at pH levels of 2.5–3.0 or below.³¹ The corrosiveness of an acid is also dependent upon the affinity of the acid for proteins. Irreversible cellular destruction results from coagulation and precipitation of proteins and the inactivation of intracellular enzymes.^{29,30} Such is the high affinity of phosphoric acid for protein that it is corrosive at pH 5.5.³⁰ Let us assume for the sake of argument that the instruments were boiled in the steriliser with a one molar strength acid. The pH of the acid would be 3 after dilution by a factor of 1000, assuming the acid is fully ionised. If the acid has a high affinity for protein (e.g. phosphoric acid), a dilution factor of 100 000 would give a pH of 5, which is still corrosive.

The final pH of the mixture administered to the patients in practice would have depended on the initial concentration of acid in the steriliser and any subsequent concentration by boiling; the amount of acid carried over into the distilled water on the inside and outside of the drawing-up needle, spinal needle, barrel and plunger of the syringe; the volume of distilled water and the efficiency with which the instruments were then washed (if at all); and finally, the quantity of acidic fluid that remained in or on the instruments when they were placed on the spinal trolley. The acid would then have been diluted up to 10 ml with cinchocaine.

Cerebrospinal fluid is a poor buffer³² and intrathecal injection would not have resulted in any appreciable decrease in acidity. Substances injected intrathecally are not metabolised locally. Elimination is effected slowly by diffusion into the epidural space and by vascular absorption.³³ Vascular thrombosis is one of the first consequences of acidic damage,³⁴ so elimination may be delayed even further. This may therefore prolong 'tissue contact time' with the acid, which would aggravate corrosive damage.³⁴

Acid contamination is supported by the following three clinical observations. Cecil Roe developed a circular ulcer surrounding the point of the spinal needle insertion, which implies that the outside of the needle was badly contaminated. The ulcer enlarged and was at least 15 cm in

diameter at the time of his death. It also eroded through to underlying muscle (personal communication, family of Mr Cecil Roe). Initially, caustic damage from a solution with a high pH was considered, since these substances are available widely as cleaning materials. However, this ulceration could also have arisen from an acidic corrosive.³⁴

Secondly, the severe pain in Cecil Roe's head and back is compatible with an injection of a strongly acidic solution,^{34,35} although presumably other substances could also be painful. Thirdly, the quality of analgesia during Cecil Roe's operation was poor. Before a local anaesthetic can be effective, it must be unionised in order to pass through the sheath that surrounds the nerve axon.³⁶ A strongly acidic environment would make cinchocaine less effective by increasing the proportion which was ionised. This is supported clinically by the observation that spinal anaesthetic drugs display tachyphylaxis when pH in cerebrospinal fluid decreases during continuous spinal anaesthesia.³⁷ Albert Woolley probably received a smaller dose of contaminant, since he suffered no symptoms of chemical irritation, was less badly affected afterwards and apparently adequately anaesthetised.

Toxicology

The first effects of acid poisoning are vascular thrombosis and secondary cellular ischaemia with a direct toxic action on cells as a result of protein coagulation and disruption of cellular enzymes.^{29,30,34} An acute myelopathy would be anticipated if a sufficient amount of a strong mineral acid had been injected intrathecally into the lumbar region. This is precisely what occurred and later the two patients began to make a partial recovery when they started to walk again with help. We have to assume, because of this improvement, that the cells and nerve fibres of the cord and cauda equina underwent some regeneration. This perhaps would be expected if the toxic action of the acid had only been partial. It is also known from other situations that the cells of the cord and the roots of the cauda equina may recover partly from an ischaemic episode.³⁸

The final and most sinister complication of injury by acid is the ultimate formation of dense fibrous tissue. This begins 2–4 weeks after the initial exposure and can be relentlessly progressive.³⁴ It is likely that, in common with other toxic substances, the meninges would be affected most severely.¹³ The cord becomes compressed and ischaemic when the meninges are constricted by fibrosis, and the patient's symptoms would then change from gradual improvement to greater weakness and spasticity. Nerve roots would become ensnared in thickened connective tissue causing root pain and muscle spasms. This condition, otherwise known as 'chronic adhesive arachnoiditis', may be complicated further by the presence of arachnoid cysts.³⁹ These were present in Cecil Roe and aggravated spasticity by additional cord compression.

Pathology

The tissues are now no longer available for examination, but the case of Cecil Roe was included in a review of neuropathological effects of intrathecal injection of toxic substances by Wolman,⁷ who was based in Sheffield in 1966. In reviewing this case the age of the patient was given wrongly but all other details accord with known facts. He

found degeneration of ascending tracts in the cervical cord with additional findings of peripheral loss of myelin from the cord in this region. There were two loculated thecal cysts from T₁ to T₁₁ formed by fibrous tissue of thickened leptomeninges. Two glial-lined syringomyelic cavities were present from T₁ in the posterior horns of the grey matter that increased in size at T₃ and extended into the anterior horns. The cavity had enlarged at T₃ and was in continuity with a segment of cord where there was no neural tissue; the cord was reduced to an organised gliotic cyst. The lumbosacral cord was reduced to a dense fibrous tag surrounded by gliotic leptomeninges. There was thickening of the pia-arachnoid with patchy loss of myelin from lower cervical nerve roots. Vascular changes were present in the meningeal vessels of the lumbosacral, thoracic and lower cervical areas. These changes included fragmentation of the elastic lamina of meningeal arteries, medial fibrosis and intimal hyperplasia.

It is apparent that the changes described in the cord by the original pathologist and reviewed by Wolman were those of extensive organisation and repair such as would follow severe and complete necrosis of part of the spinal cord. It is equally apparent that there was a severe fibrotic reaction in the meninges that corresponded to a chronic adhesive arachnoiditis. Wolman concluded that it was likely that the arachnoiditis and thoracic arachnoid cysts were the result of a contaminant of the spinal anaesthetic agent, since these were seen during laminectomy before the administration of intrathecal alcohol. The nerve root myelin loss and demyelination of the cervical cord were, in his opinion, consistent with the known effects of the later alcohol injections.

It is important to consider whether these changes of organised extensive necrosis of the cord are consistent with the effects of injection of phenol as a contaminant of the local anaesthetic, as suggested in subsequent legal proceedings in the case of Roe. Intrathecal phenol injections were used widely as a method of pain relief and there were detailed neuropathological follow-up of patients treated in this way. Four patients studied at postmortem examination after intrathecal injection of 7.5% or 10% phenol showed changes of demyelination of nerve roots only.²¹ Pathological studies on 19 patients treated with intrathecal phenol injection⁴⁰ showed that such treatment caused degeneration of nerve fibres in nerve roots, but was associated only with secondary degeneration of fibres in the posterior columns of the spinal cord; there was no evidence of excessive pathological reaction to such treatment, with the exception of one person who received an injection of silver nitrate in phenol and glycerol and developed meningitis. These findings were supported by other similar studies.⁴⁰ There is thus a weight of neuropathological evidence which is against the concept that the extensive damage to the cord seen in the case of Roe can be attributed to intrathecal phenol injection.

It is pertinent to consider the likely sequelae of contamination by a strong mineral acid. The immediate toxic reactions of mineral acids are related to protein coagulation in cells, resulting in cell death. Vascular thrombosis occurs in affected vessels. It is likely that injection of acid in the thecal sac would result in protein coagulation in exposed vessels with secondary thrombosis and resulting spinal cord infarction. Inflammatory necrosis produced in the leptomeninges is likely to have excited an acute inflam-

matory reaction, followed by the development of fibrosis. The postmortem finding of vessels in the arachnoid with old intimal proliferation and elastic lamina fragmentation is consistent with such a secondary vascular pathology. Segmental necrosis of the spinal cord would have undergone organisation and repair to form the gliotic cyst encountered at postmortem, and it is not unusual for syringomyelic cavities to occur above the level of such organised damage.³⁹ The findings here bear a close similarity to the clinicopathological syndrome of 'chronic adhesive arachnoiditis' associated with spinal anaesthesia, and described by Greene.¹³

In conclusion, the postmortem findings in the case of Roe are consistent with the intrathecal injection of a mineral acid which may have contaminated the spinal anaesthetic.

Sequence of events

It must be stressed that the following observations are partly conjectural, but do represent what appears to be the likely sequence of events on that Monday and the preceding weekend.

Three facts help to confirm the steriliser as the source of acid contamination. All three cases occurred on a Monday. Cecil Roe, the first patient on the operating list, was affected more seriously than Albert Woolley. Finally, these three incidents were limited to one day only, with no recurrence of the problem. The steriliser would have been contaminated with acid on the Monday morning, if, as part of routine weekend maintenance, it were descaled and the person responsible had forgotten to drain and wash out the acid, or perhaps had done this inadequately. The needles and syringes would have been contaminated when they came to be used by the anaesthetist, if, on the Monday morning, the acidic solution were mistaken for ordinary water. The initial effect of boiling is to concentrate the acidic solution. However, throughout the day, as evaporated acid solution was replaced regularly by fresh water, there would be a gradual decline in acidic strength. This change in pH probably explains why Albert Woolley, the third patient, received a smaller dose of contaminant. It was standard procedure to drain sterilisers at the end of the day, so the acidic solution would have been removed on the Monday evening, and no further problems could have arisen.

Conclusion

The diagnostic criteria which are required to establish that neurological damage is a direct consequence of a spinal anaesthetic were described by Greene.¹³ There is no doubt that in the case of Woolley and Roe, these criteria have been satisfied and that the causative agent was most probably a mineral acid.

Some of the early episodes of arachnoiditis after spinal anaesthesia have never been adequately clarified. Acid from a steriliser could have been the source of some of these problems. The ease with which contamination could happen, and the relatively small volumes of acid that would have been required, make this a realistic possibility. There is a very close similarity between the pathological and clinical features of these two cases and those which arose before.^{13,41} This is very suggestive of a common aetiology,

although myelopathy and arachnoiditis can result from a wide range of other causes.^{13,39} However, the erratic occurrence¹³ of these early anaesthetic injuries in both time and place is very suggestive of a complication dependent on unpredictable human error. In addition, the decrease in the incidence of postspinal paralysis¹³ in the late 1950s may be associated with the introduction of autoclaving; the use of the water boiling steriliser was then abandoned.

The impetus to find a solution to this problem would have diminished in the 1950s and 1960s as the incidence of paralysees decreased. However, we must now ask why an answer was not found in the 30 years or so preceding this. One factor was the gulf between the working environments of the maintenance and medical staff. The maintenance staff who were responsible for descaling were unlikely to have been aware of a surgical patient who was found later to be suffering severe neurological problems. The medical staff in their turn were not associated in any way with cleaning and maintenance duties. It is not surprising that the connexion was never made. It became apparent at the trial that if there had been severe neurological sequelae after spinal anaesthesia, the neurologist did not consider it necessary to inform the anaesthetist about this.²⁵ More discussion between those concerned might have directed their thoughts in the right direction.

It is ironic, but also very fitting, that Professor Macintosh, who advised autoclaving spinal instruments to avoid hypothetical seepage of immersion fluid as well as to provide more reliable sterility, had also quite unwittingly prevented any further repetition of the events of the very case in which he had been so closely involved.

Acknowledgments

The author thanks the family of Mr Cecil Roe for their help and cooperation. Invaluable assistance was provided by Dr M.A. Healy, Senior Lecturer in Chemistry, University of Nottingham, Dr J.S. Lowe, Senior Lecturer and Consultant in Neuropathology and Dr A. Whiteley, Consultant Neurologist, Queen's Medical Centre, Nottingham. The author is also grateful for help from the staff of the North Derbyshire Health Authority and Nottingham Health Authority. Finally, the author would like to thank Professor A.R. Aitkenhead, University Department of Anaesthesia, Queen's Medical Centre, Nottingham, for his advice on the preparation of the manuscript.

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Forum

Total intravenous anaesthesia with propofol and buprenorphine

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Summary

A combination of propofol infusion and two bolus doses of buprenorphine, 2.5 or 5.0 µg/kg were evaluated in a total intravenous anaesthesia technique in 36 patients of ASA grade 1 or 2 undergoing cholecystectomy. Additional boluses of propofol were given intravenously if needed. Systolic blood pressure after tracheal intubation increased significantly only in those who received the smaller dose of buprenorphine. Patients in both groups remained haemodynamically stable throughout surgery with minimal side effects. Recovery was fast even with prolonged infusions and without major side effects. No patient reported awareness on postoperative questioning.

Key words:

*Analgesic narcotic; buprenorphine.
Anaesthetic intravenous; propofol.*

Propofol may be given by bolus injection or continuous infusion^{1,2} and is a suitable agent for total intravenous anaesthesia. Research has focused on the advantages of total intravenous anaesthesia, a method which avoids atmospheric pollution,^{3,4} the need for vaporizers⁵ and eliminates problems associated with nitrous oxide anaesthesia in middle ear disease,⁶ gut surgery,⁷ and its effect on the nervous and haemopoietic systems.⁸ It has an added advantage in war situations⁹ and in parts of the Third World where medical gases may be unavailable.

Propofol possesses many of the properties required for total intravenous anaesthesia,^{10,11} particularly a short recovery time in spite of evidence of accumulation in long procedures.¹² The present study was designed to evaluate the use of propofol with one of two doses of buprenorphine as a method of total intravenous anaesthesia.

Methods

The study was approved by the hospital Ethics Committee. Male or female patients classified as ASA 1 and 2 aged between 16 and 60 years scheduled for cholecystectomy were studied. Informed patient consent was obtained and the patients were randomly allocated into two groups of 18 each. None of the patients had clinical or suspected hepatic disease. Patients in both groups were premedicated with diazepam 0.15 mg/kg orally 2 hours before operation.

On arrival in the operating room an 18-gauge intravenous cannula was inserted in a large hand vein. Baseline measurements of systolic, diastolic and mean arterial blood pressure were obtained using a noninvasive monitor (Datex). Continuous monitoring of CM5 lead of the ECG, and oxygen saturation (Ohmeda 1400) were established.

An anaesthetist unconnected with the study gave a bolus of intravenous buprenorphine so that the observer was blinded to the dose received. Group A patients received 2.5 µg/kg and group B 5.0 µg/kg. The propofol dose regimen was that described by Roberts *et al.*¹³ and was the same for both the groups. An initial dose of 1 mg/kg was given over 20 seconds followed by an infusion of 10 (mg/kg)/hour for the first 10 minutes. Pancuronium 0.1 mg/kg was injected after the loss of eyelash reflex. The patients' lungs were ventilated via a mask and Magill system with oxygen-enriched air (F_{IO_2} 0.4) to normocapnia and the trachea was intubated 3 minutes after the injection of pancuronium. Propofol infusion was continued at a rate of 8 (mg/kg)/hour for the next 10 minutes and then reduced to 6 (mg/kg)/hour for the duration of surgery. The infusion was discontinued at the time of the last skin stitch.

The patients' lungs were ventilated throughout with oxygen-enriched air using a Manley Servoventilator. The $P_{ET}CO_2$ was kept between 0.05 and 0.055. Muscle relaxation was maintained with incremental doses of pancuronium as judged by a nerve stimulator. Stability and depth of anaesthesia were evaluated with regard to need for additional bolus doses of propofol 10 mg. The criteria used to judge the depth of anaesthesia were the presence of two out of four signs: increase in systolic arterial blood pressure 15% above baseline; increase in heart rate 15% above baseline; lacrimation; and sweating. The blood pressure was monitored one minute after buprenorphine injection, 2 minutes after induction with propofol, every minute after tracheal intubation for 3 minutes and every 5 minutes thereafter. ECG, oxygen saturation and nasopharyngeal temperature were displayed continuously. Visual monitoring of train-of-four stimulus was charted every 15 minutes. The residual

Accepted 6 October 1989.

Table 1. Patient data. Values expressed as mean (SD) or number of patients.

	Group A (propofol/ buprenorphine 2.5 µg/kg) (n = 18)	Group B (propofol/ buprenorphine 5 µg/kg) (n = 18)
Age; years	40.88 (10.61)	41.22 (12.06)
Weight; kg	59.35 (14.34)	59.33 (8.05)
Males/females	1/17	2/16
ASA, grade 1/2	4/14	6/12
Pre-induction systolic arterial blood pressure; mmHg	137.44 (16.98)	142.89 (16.99)
Pre-induction heart rate; beats/minute	89.38 (17.02)	91.83 (15.95)
Duration of anaesthesia; minutes	139.88 (61.82)	166.44 (64.02)

There was no significant difference between the groups (Student's *t*-test).

neuromuscular blockade at the end of surgery was reversed with neostigmine. The time from cessation of propofol infusion until the patients opened their eyes to command and the interval before the correct date of birth given were recorded.

An overall assessment of the quality of anaesthesia was made by the anaesthetist in charge of the case.

Statistical analysis. The demographic details and duration of anaesthesia were compared using Student's *t*-test. Arterial blood pressure and heart rate data were subjected to analysis of variance applied to each time point. The incidence of side effects within the two groups was compared by Chi-squared test with Yates' correction. Values of $p < 0.05$ were considered to indicate statistical significance.

Results

Thirty-six patients (three males and 33 females) aged between 24 and 64 years who weighed between 35 and 88 kg were studied. The demographic data are given in Table 1. There was no significant difference between the groups with regard to weight, age, distribution of males and females, ASA classification, pre-operative blood pressures, pre-operative pulse rate, duration of infusions and duration of anaesthesia.

Complications during induction are shown in Table 2. The highest incidence was that of pain on injection of propofol bolus. The frequency of this complaint was higher in group A.

The changes in the systolic blood pressure associated with the various stages of the procedure are shown in Figure 1. The blood pressure remained stable after buprenorphine injections in both groups, but after propofol it decreased slightly in both groups. Group B showed a greater percentage decrease compared to baseline values, 7% compared to 1.3%. Both groups showed an increase above baseline values after tracheal intubation, but the increase was statistically significant only in group A, 3 minutes after intubation. This pressor response did not correlate with age, sex or ASA grade. The systolic blood pressures then returned to below the baseline values in both groups. The pressures recorded one minute after incision in group B showed a significant decrease compared to baseline values (-12%). The blood pressures then remained stable throughout the rest of the procedure. No statistical difference was observed between the groups at any stage.

The changes in the heart rate are shown in Figure 2. The heart rate remained stable after induction in both groups. Tracheal intubation produced a significantly greater increase compared to the baseline in group A patients, where the pulse rate remained significantly elevated until

Table 2. Side effects observed in the two groups.

	Group A (propofol/ buprenorphine 2.5 µg/kg) (n = 18)	Group B (propofol/ buprenorphine 5 µg/kg) (n = 18)
Induction		
Pain on injection	8	3
Involuntary movements	2	2
Skin rash	0	1
Tachycardia (beta blockers given for control)	1	0
Bronchospasm	1	0
Maintenance		
Bronchospasm	1	0
Movements not related to light anaesthesia	1	0
Recovery		
Nausea	1	2
Confusion and restlessness	2	0
Vomiting	0	1
Bradycardia (heart rate < 60/minute)	0	1

There was no significant difference between the groups (Chi-Squared test with Yates' correction).

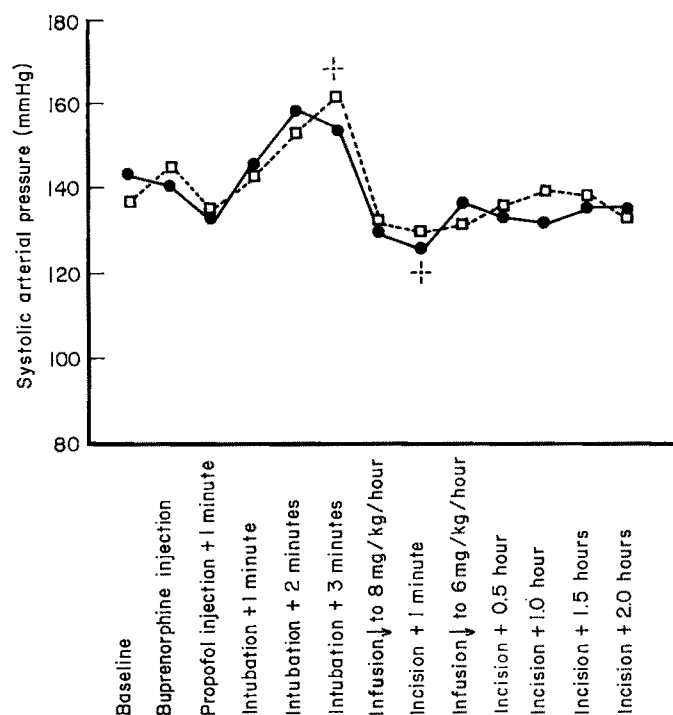


Fig. 1. Mean systolic arterial pressure (mmHg) during propofol infusion + buprenorphine 2.5 µg/kg (□) compared with propofol infusion + buprenorphine 5 µg/kg (●) at indicated stages. \ddagger = $p < 0.05$ statistically significant difference from baseline value.

after the incision and did not return to control values. In group B there were clinically similar changes in the heart rate after tracheal intubation, but the percentage increase in heart rate was not statistically significant. There was no statistical difference observed between the two groups. One patient in group A had a sinus tachycardia immediately after the bolus of propofol and a β -adrenoceptor blocker

(propranolol) was necessary. None of the patients had sinus bradycardia (< 60 beats/minute) intra-operatively.

The range and the mean dosages of propofol used in the two groups during induction and maintenance are shown in Table 3. Total induction dose was calculated as the (bolus dose + the initial fast phase infusion rates of 10 (mg/kg)/hour for 10 minutes and 8 (mg/kg)/hour for the next 10

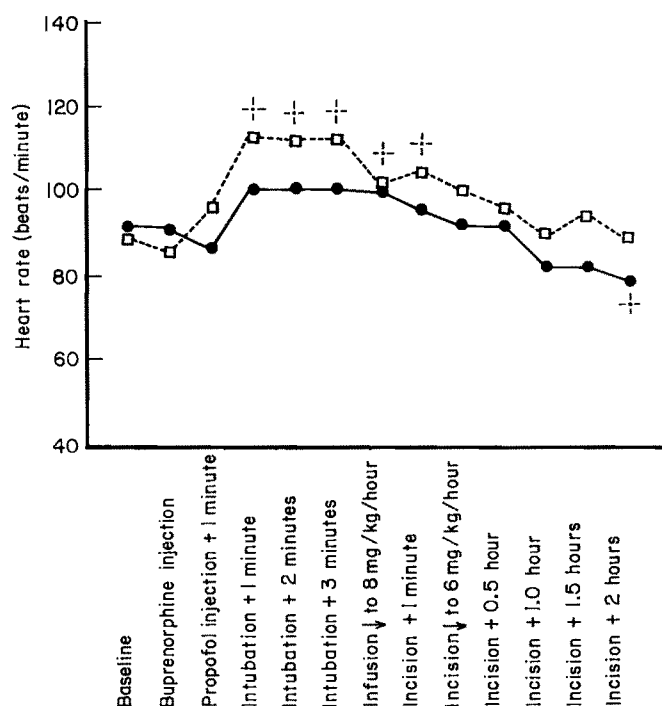


Fig. 2. Mean heart rate changes (beats/minute) during propofol infusion + buprenorphine 2.5 µg/kg (□) compared with propofol infusion + buprenorphine 5 µg/kg (●) at indicated stages. \ddagger = $p < 0.05$ statistically significant difference from baseline value.

Table 3. Mean (SD) propofol dosages in both groups.

	Group A (propofol/ buprenorphine 2.5 µg/kg) (n = 18)	Group B (propofol/ buprenorphine 5 µg/kg) (n = 18)
Duration of infusion; minutes	135.88 (62.66)	157.61 (65.46)
Range; minutes	80–326	86–325
Total mean induction dose of propofol; mg	236.47 (57.42)	235.95 (35.09)
Total mean maintenance dose of propofol; mg	704.47 (434.03)	846.66 (443.5)
Range of total dose of propofol used; mg	433–1763	531–2080
Mean rate of utilisation during maintenance; (mg/kg)/hour	6.15	6.24
Mean rate of utilisation during induction and maintenance; (mg/kg)/hour	6.91	7.093

There was no significant difference between the groups.

minutes). Total maintenance dose was calculated at 6 (mg/kg)/hour for the duration of infusion. The mean rate of utilisation during induction and maintenance was calculated as the total dose (including induction dose) divided by the duration of infusion. The mean rate of utilisation during maintenance was calculated by the total dose minus (induction dose + initial fast phase infusion) divided by the duration of infusion. No significant difference was observed between the groups as regards their mean rate of utilisation, the total induction dose and the maintenance dose. It was necessary to administer supplementary boluses of propofol in one patient in group A and four in group B.

The incidence of side effects observed in the maintenance period are given in Table 2. Table 4 shows the recovery data. The indices of immediate recovery studied were times from end of infusion to opening eyes to command and the ability to give name and date of birth correctly. The mean time to opening eyes to command was 10.38 (SD 4.23) minutes in group A and 11 (SD 3.75) minutes in group B. No significant difference was observed between the two groups in either indices of recovery.

No correlation was observed between the duration of propofol infusion and opening eyes to command after termination of infusion (Fig. 3). The overall incidence of untoward effects observed in recovery remained low in both groups (Table 2). Postoperative nausea occurred in only one patient in group A and two in group B. One patient in group B vomited.

Two hours after the end of surgery the patients were specifically asked about awareness during anaesthesia. None of the patients reported any awareness. The anaesthetic was rated either good or satisfactory by the consultant anaesthetist involved with the case in all the patients in both groups.

Discussion

The main objective of our study was to develop a technique of total intravenous anaesthesia which would provide satis-

factory anaesthetic conditions for major abdominal surgery. An infusion of propofol was shown to provide satisfactory anaesthesia with nitrous oxide-oxygen mixtures in both spontaneously breathing patients or combined with a muscle relaxant,^{14,15} but little work has been done on total intravenous techniques using propofol infusions combined with air-oxygen mixtures and narcotic drugs, especially in major abdominal surgery,^{16,17} although Steegers and Foster have used the method for orthopaedic surgery.¹⁸ There is also no published work on the effect of buprenorphine on the quality of anaesthesia with propofol in major abdominal surgery.

No marked hypotension was seen after the bolus injection of propofol. Other studies have commented on the hypotensive effect of 2 mg/kg bolus doses due to a decrease in systemic vascular resistance in healthy subjects.² Our results are in agreement with the studies using 1 mg/kg as induction dose.¹³ Buprenorphine was shown to be a suitable narcotic for use in major abdominal surgery, since it is associated with cardiovascular stability; it was shown to attenuate the hypertensive response to laryngoscopy and intubation in adult patients if given before induction. Propofol also attenuates the haemodynamic effect of laryngoscopy and intubation,^{19–21} and the combination of two may result in a smoother induction. This effect was observed in our study especially with buprenorphine 5 µg/kg. The heart rates remained fairly stable in group B but increased significantly above baseline in group A patients.

The commonest side effect observed at induction was pain on injection. Eight of the patients in group A and three in group B complained of pain. The fewer number who complained in group B may be because of the analgesic effect of 5 µg/kg buprenorphine given before induction. One patient with a history of asthma developed severe bronchospasm soon after injection of propofol. This was treated with injection of aminophylline 125 mg intravenously followed by an infusion of 250 mg in 500 ml dextrose for the next 3 hours.

One ASA 2 patient aged 28 years, with a baseline blood

Table 4. Mean (SD) recovery from end of propofol infusion.

	Group A (propofol infusion/ buprenorphine 2.5 µg/kg) (n = 18)	Group B (propofol infusion/ buprenorphine 5 µg/kg) (n = 18)
Time to opening eyes on command from end of infusion; minutes	10.38 (4.23)	11.0 (3.75)
Time to orientation from end of infusion; minutes	18.37 (9.34)	25.5 (14.9)

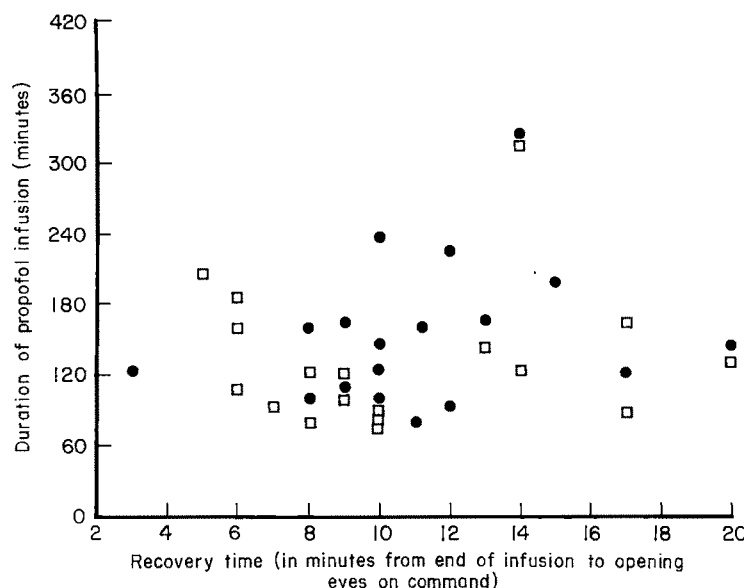


Fig. 3. Recovery time (in minutes from termination of infusion to opening eyes on command) plotted against duration of propofol infusion. Symbols as for Figs 1 and 2.

pressure of 129/83 and heart rate of 117/minute, developed a sinus tachycardia of 180 beats/minute immediately after the injection of propofol and responded to intravenous propranolol.

The continuous infusion of propofol with buprenorphine produced stable anaesthesia. The total dose of propofol and the average infusion rates did not vary significantly between the groups. The patients with the smaller dose of buprenorphine did not require additional supplementation with propofol. However, patients in group B did show more cardiovascular stability. We found that the assessment of depth of anaesthesia according to the four criteria described were reliable; only five patients required bolus doses of propofol, one in group A and four in group B.

Recovery was rapid in both groups and there was a low incidence of side effects. Three patients in group A and three in group B had infusions lasting for more than 3 hours, but awoke within 15 minutes of discontinuation of infusion. None of the patients reported awareness when questioned postoperatively.

In conclusion, the data in our study suggest that propofol is a suitable agent for continuous infusion in total intravenous anaesthesia, with buprenorphine as the analgesic component in paralysed patients undergoing abdominal surgery. We recommend a 5.0 µg/kg bolus dose of buprenorphine because of its greater haemodynamic stability when combined with propofol.

Acknowledgment

The authors are grateful to ICI Pharmaceutical (Pakistan) Limited for supplies of Propofol.

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Anaesthesia, 1990, Volume 45, pages 870-872

Oral premedication in children

A comparison of trimeprazine with a trimeprazine, droperidol and methadone mixture

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Summary

One hundred children who presented for minor general surgical procedures were randomly assigned to receive one of two oral premedications. Those in group A ($n = 50$) were given 3 mg/kg of trimeprazine and those in group B ($n = 50$) a mixture of trimeprazine 1.0 mg/kg, droperidol 0.15 mg/kg and methadone 0.08 mg/kg. Patients in group B were more likely to be asleep on arrival in the anaesthetic room ($p < 0.02$) and were less likely to be distressed at induction of anaesthesia ($p < 0.02$). Thiopentone requirements were less in group B ($p < 0.001$). The incidence of side effects was similar in the two groups. It is concluded that the mixture produces more satisfactory sedation than trimeprazine.

Key words

Anaesthesia; paediatric, premedication.
Premedication; trimeprazine, droperidol, methadone.

Trimeprazine tartrate syrup has become a standard paediatric premedication in British hospitals. Oral sedation is more acceptable to children than an intramuscular injection and it has been shown to provide good sedation in most cases.¹⁻⁶ However, the effect of trimeprazine on individual children given the same mg/kg dose can vary greatly. Some children arrive in the anaesthetic room wide awake while others are deeply sedated.

Other drugs have been added to the trimeprazine syrup in an effort to provide more reliable sedation. It was shown that droperidol and trimeprazine combinations are superior to trimeprazine alone; sedation is more uniform, postoperative vomiting less frequent and analgesic requirements are reduced.⁷ In contrast, a comparison of trimeprazine with a trimeprazine, droperidol and methadone mixture, failed to show any difference in sedation.⁸

A mixture of trimeprazine, droperidol and methadone (known as TDP) has been in use in this hospital for some time. We have found it to give more reliable sedation than trimeprazine. We compared these two premedications in a randomised double-blind study in order to determine if this impression was correct.

Methods

Ethics committee approval was given and informed consent obtained from the parents of children enrolled in the study.

One hundred healthy children presenting for minor surgical procedures were included. They were randomly allocated into trial groups A or B to receive one of two raspberry-flavoured syrups that were made up by the hospital pharmacy. One syrup contained trimeprazine 12 mg/ml (group A); the other syrup TDP (trimeprazine 4 mg/ml, droperidol 0.6 mg/ml and methadone 0.32 mg/ml; group B). Each child received 0.25 ml/kg of the appropriate mixture. This was equivalent to 3 mg/kg in the trimeprazine-only group and 1 mg/kg of trimeprazine combined with 0.15 mg/kg of droperidol and 0.08 mg/kg of methadone in the other group.

Syrup A or B was given some 2.5 hours before induction of anaesthesia so that sedation was present when the patients were transferred with their parents to a children's reception area adjacent to the operating theatre. All patients had EMLA cream applied to the back of both hands at the time that the premedication was given.

On arrival in the anaesthetic room patients were recorded as being either asleep or awake by an anaesthetist who was unaware of the premedication given. If awake it was noted if they were calm or distressed. Anaesthesia was induced, after insertion of a 23-gauge intravenous cannula, with thiopentone 5 mg/kg given over 10 seconds. Supplemental increments of 1 mg/kg were given as required using loss of eyelash reflex to indicate adequate depth of anaesthesia. The behaviour of the child at induction was noted as

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Accepted 29 March 1990.

Table 1. Details of patients and operations performed. Values are expressed as mean (SD).

	Group A (n = 50)	Group B (n = 50)
<i>Patient details</i>		
Age; years	5.4 (2.5)	6.3 (1.8)
Weight; kg	20.6 (6.7)	21.4 (6.9)
Males	47	45
Females	3	5
<i>Operations performed</i>		
Circumcision	27	28
Orchidopexy	10	10
Hernia repair	10	9
Others	3	3
Duration of surgery; minutes	31 (10.7)	31 (11.4)

either calm and cooperative or uncooperative and (or) distressed. Anaesthesia was maintained with enflurane 1–2% in nitrous oxide/oxygen.

All patients were given a caudal block for postoperative analgesia. The volume of bupivacaine 0.25% administered was determined by the patient's weight and the surgical procedure.⁹ The block was initiated in the anaesthetic room after induction of anaesthesia and before surgery commenced. Duration of surgery was taken as the time from induction of anaesthesia to the placing of the last skin suture.

Postoperative assessment commenced in the recovery ward; the assessors were blind to the premedication given. Restlessness, nausea and vomiting, pallor and the need for further analgesia was noted by the recovery nurse. Recovery time was taken as the time from entry to the recovery area to the time of airway rejection. Similar observations were taken on the ward in the 24 hours after surgery, and details of side effects and analgesic requirements were recorded.

All data were subjected to statistical analysis by Student's *t*-test or Chi-squared tests as appropriate. A probability value of less than 0.05 was taken to represent statistical significance.

Results

The two groups were similar in age, weight, and sex distribution and in the duration and type of surgery carried out. The majority of the patients underwent circumcision, orchidopexy or hernia repair (Table 1).

Both groups of patients received their premedication at similar times before induction of anaesthesia. Those who received TDP (group B) were significantly more likely to arrive in the anaesthetic room asleep ($p < 0.02$). Fewer patients in this group were distressed on arrival, but this difference was not statistically significant. Significantly fewer patients in group B were distressed or uncooperative at the time of cannula insertion and significantly fewer patients in this group required additional thiopentone during induction of anaesthesia ($p < 0.02$ and < 0.001 respectively) (Table 2).

In the recovery area there was no difference in the incidence of side effects in the two groups nor was there any difference in the numbers of patients who required additional analgesia. This number was low and reflected the use of caudal analgesia. Time to rejection of the airway was slightly longer in group B (Table 3).

There was no difference in the analgesic requirements of the two groups over the 24 hours after surgery. The incidence of side effects on the ward was also similar. Seven

Table 2. Arrival in anaesthetic room and induction. Values are expressed as mean (SD).

	Group A (n = 50)	Group B (n = 50)
Time of premedication before induction; minutes	157 (34)	160 (26)
<i>Condition on arrival</i>		
Asleep	12	24*
Awake, calm	29	23
Awake, distressed	9	3
<i>Response to induction</i>		
Distressed/uncooperative	12	3*
Extra thiopentone given	21	3*

*Statistically significant.

patients in group A and five in group B had mild nausea and vomiting. Five patients in each group were noted to be distressed.

Discussion

There is some debate as to what is the best approach to use in the premedication of young children who present for elective surgery. Some anaesthetists rely on the pre-operative visit to establish a rapport with the patient and avoid the use of premedicant drugs altogether. This offers the benefit of rapid recovery and enables discharge early in the postoperative period. However, with some children, particularly the very young, it is often difficult to establish a good relationship during the brief pre-operative visit and it is our experience that many children, while they appear relaxed and cooperative on the ward, behave quite differently in the strange surroundings of the anaesthetic room. Consequently we believe that there will always be a need for sedative premedication in a proportion of cases.

The results of this study demonstrate that it is possible to improve upon the quality of sedation provided by trimeprazine tartrate. The use of TDP produces a child who is more reliably sedated on arrival in anaesthetic room. This, together with the use of EMLA cream to facilitate painless venous cannulation, allows the anaesthetist to achieve a smooth induction of anaesthesia.

Fewer patients in the TDP group required additional thiopentone at induction. This finding supports previous work, that sedation with TDP reduces the thiopentone dosage required for induction of anaesthesia.¹⁰

There was no increase in the incidence of side effects when this combination of drugs was used, despite the improvement in sedation, and the time to recovery was

Table 3. Behaviour in recovery room. Values are expressed as mean (SD).

	Group A (n = 50)	Group B (n = 50)
Time to airway rejection; minutes	21 (11)	28 (16)*
Patients who required additional analgesia	5	2
<i>Side effects</i>		
Vomiting	0	1
Pallor	6	4
Restlessness	11	5

*Statistically significant.

only slightly prolonged. A previous study was unable to demonstrate an improvement in sedation when TDP was used, despite a similar method of assessment.⁸ The adequacy of sedation in our study was tested by a higher disturbance factor, in that children were moved from a distant children's ward to the noisy environment of the operating theatre. A quiet room was provided in the previous study in close proximity to the anaesthetic room and the disturbance to the children was less. This may explain why the previous workers failed to show any difference between the two sedative regimens. Another factor may be that lower doses of trimeprazine were given to both groups in the current study.

It may be that TDP will provide useful analgesia during the recovery period. It has, however, been our standard practice to provide caudal analgesia to patients who have this type of surgery and we believed that although this may mask any potential analgesia benefits of TDP, it would be unethical to change our practice for this study.

Acknowledgments

The authors thank Mrs C. Evans, medical secretary and The Department of Medical illustration for preparing the manuscript, Mr K. D. Vellacott, FRCS, Consultant Surgeon, for his support and cooperation, Mr G. V. Brangham, MR Pharm S, Pharmacy Manager, Mr G. Titley, BA, ALA, Librarian, Royal Gwent Hospital and

the many ward and theatre staff whose hard work made this study possible.

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Anaesthesia, 1990, Volume 45, pages 872-875

Anaesthesia for cardioversion

A comparison between propofol, thiopentone and midazolam

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Summary

This study compares the induction and recovery characteristics, haemodynamic changes and side effects of propofol, thiopentone and midazolam when used as the anaesthetic agents for cardioversion. Recovery after midazolam was significantly longer ($p < 0.05$) than with either thiopentone or propofol. There was no difference in the recovery times between thiopentone and propofol. There was a significant decrease in mean arterial pressure 2 minutes after induction with propofol and midazolam. Three patients each in the thiopentone and propofol groups needed assisted ventilation because of apnoea, and four patients each in the propofol and midazolam groups had low SpO_2 values ($< 95\%$). Flumazenil was used to reverse the effects of midazolam in eight patients and five of these were still drowsy 4 hours after the procedure. This study indicates that thiopentone is the most satisfactory agent for anaesthesia for cardioversion.

Key words:

Anaesthetics, intravenous; thiopentone, propofol. Hypnotics, benzodiazepines; midazolam.

Direct current countershock after its first introduction in 1962¹ is used commonly today for the conversion of abnormal cardiac rhythm to normal sinus rhythm. Many different anaesthetic techniques were described for this

purpose; the agent used most commonly is thiopentone. Muenster and his colleagues² reported a greater frequency of extrasystoles after thiopentone as compared to the number after diazepam, which then became an alternative

Table 1. Patient characteristics of the three groups studied. Values are expressed as mean (SD).

	Propofol (10)	Thiopentone (10)	Midazolam (10)
Age; years	64 (6)	66 (7)	70 (6)
Sex; M/F	5/5	9/1	8/2
Weight; kg	74 (10)	74 (11)	76 (8)
Rhythm; atrial fibrillation, atrial flutter	8/2	9/1	10/0
Successful cardioversions	10/10	9/10	8/10

method for induction of anaesthesia in these patients.³⁻⁵ However, many side effects associated with the use of diazepam are reported including unpredictability of effect,⁶ high incidence of recall^{7,8} and ventricular arrhythmias.⁹

Midazolam sedation is also used and is reported to be effective for cardioversion.^{10,11} Midazolam, according to Kaplan,¹² is ideal for short procedures such as cardioversion. Propofol was recently used as an induction agent for cardioversion¹³ and shown to have shorter recovery times compared to thiopentone.

The aim of this study was to compare propofol, thiopentone and midazolam as induction agents for elective cardioversion and to investigate the recovery characteristics, haemodynamic changes and side effects associated with these drugs.

Methods

The study was approved by the Hospital Ethics Committee and verbal consent was taken from all patients before the study. Thirty unpremedicated patients (ASA groups 2-3), were randomly allocated to receive either propofol, midazolam or thiopentone in such a way that there were 10 patients in each group. Digoxin was discontinued 24 hours before the procedure and all patients were fasting for at least 6 hours. A Nellcor N-200 pulse oximeter was used continuously to display SpO_2 after the procedure was explained to the patient. Arterial blood pressure was measured every minute using an automatic device (EME Auto BP, Brighton, UK). An intravenous cannula inserted in the left arm was used for injecting drugs and taking blood samples as necessary.

The patients' lungs were pre-oxygenated for 2-3 minutes before induction of anaesthesia until the SpO_2 reached 98%. All anaesthetics were given by the same anaesthetist; the observer had no information about the drug used. A stop-watch was started at the onset of induction of anaesthesia.

Propofol and thiopentone were injected continuously over a 1-minute period until loss of the eyelash reflex, and subsequent increments were given as needed to achieve this point. Midazolam 5 mg were injected over 1 minute, and subsequent 2-mg increments were given until loss of the eyelash reflex. The patients' lungs were artificially ventilated with 100% O_2 via a facemask if SpO_2 was less than 95% or if the patient was apnoeic for more than 30 seconds at any time during the procedure.

Cardioversion was attempted by the cardiologist using S&W-DMS 600/3 (Albertslund, Denmark) apparatus, with the paddles placed at the right upper sternal border and apex of the heart. The intensity of the shock given depended on the type of rhythm, the success of previous attempted cardioversions, and the cardiologist who performed the procedure. The continued loss of eyelash reflex was used as a guide for the need of supplement anaesthetic if more than one attempt was needed to cardiovert the patient.

The following recovery times were noted: opening eyes on command; recalling 'date of birth' on questioning; ability to do simple calculations e.g. $100-7$. All patients were interviewed 4 hours after the procedure and questioned about the side effects and awareness during the procedure.

Results are presented as mean (SD). Data were analysed by the paired Student's *t*-test, $p < 0.05$ was considered to be significant.

Results

The mean age and weight of the patients were comparable (Table 1). There were more females in the propofol group compared to the others. Propofol (2.2 (0.3) mg/kg) and midazolam (0.24 (0.03) mg/kg) were associated with a significant decrease in mean arterial blood pressure 2 minutes after induction of anaesthesia. In contrast, thiopentone (5.2 (1.0) mg/kg) did not cause a significant decrease in mean arterial blood pressure (Fig. 1). The mean blood pressure continued to be significantly lower in the propofol group during the entire period of the study.

The 'time to loss of eyelash reflex' was similar in the propofol and thiopentone groups but significantly longer in the midazolam group (Table 2).

Recovery characteristics were similar in the thiopentone and propofol groups and there was no significant difference in the three recovery times. However, patients in the midazolam group took much longer to recover (Table 3), and it was decided to wake them 15-30 minutes after induction by giving flumazenil (0.3-0.5 mg). The response to flumazenil occurred within 2-3 minutes but was short-lasting. Five of 10 patients were asleep at the time of interview 4 hours later.

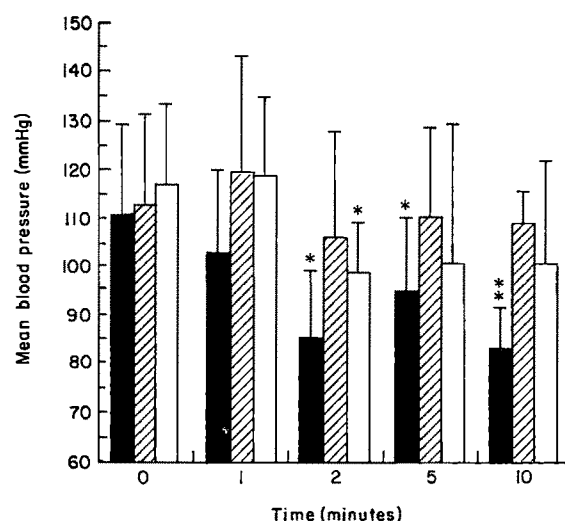


Fig. 1. Mean blood pressure, expressed as mean (SD), during cardioversion in the three groups studied. ■, propofol; ▨, thiopentone; □, midazolam. * $p < 0.05$; ** $p < 0.05$.

Table 2. Doses, ventilation and time to loss of eyelash reflex in the three groups studied. Values are expressed as mean (SD).

	Propofol (10)	Thiopentone (10)	Midazolam (10)
Total dose; mg	164 (25)	380 (85)	18.1 (3.2)
Total dose; mg/kg	2.2 (0.3)	5.2 (1.0)	0.24 (0.03)
Time to loss of eyelash reflex; seconds	100 (16)	99 (14)	339 (73)*
Assisted ventilation			
Apnoea > 30 seconds	3	3	—
SaO ₂ < 95%	4	1	4

*p < 0.05.

Table 3. Recovery characteristics in the three groups studied. Values are expressed as mean (SD).

	Propofol (10)	Thiopentone (10)	Midazolam (10)
Opening eyes on command; seconds	639 (182)	616 (253)	> 900*
Recalling date of birth; seconds	689 (172)	656 (270)	> 900*
Doing simple calculations; seconds	719 (186)	673 (266)	> 900*

*p < 0.05.

Three patients (30%), each in the propofol and thiopentone groups, required assisted ventilation because of apnoeic episodes that lasted more than 30 seconds. No patient in the midazolam group had apnoea that lasted longer than 30 seconds (Table 2). Four patients (40%) each, in the propofol and midazolam groups, had SpO₂ values < 95%, while only one patient in the thiopentone group had low SpO₂. However, all four patients in the propofol group had low SpO₂ within 10 minutes of induction of anaesthesia, while three of four patients in the midazolam group had low SpO₂ after 10 minutes.

Twenty-seven of 30 patients (90%) were successfully cardioverted.

No patient complained of nausea or vomiting after the procedure. Two patients in the thiopentone group recalled dreaming, both of which were pleasant. Five patients (50%) in the midazolam group were drowsy 4 hours after the procedure, although they could reply to all the questions put to them. Eight patients who received midazolam typically responded to cardioversion by a short cry that was associated with flexion movements of the upper and lower limbs; this persisted until the patient was restrained. However, no patient in any of the groups was aware during the procedure or recalled having pain during cardioversion.

Discussion

The recovery times in this study suggest that there is no significant difference between propofol and thiopentone. This is in contrast to earlier studies¹³⁻¹⁵ that have shown that propofol has a shorter recovery time than thiopentone. This could be explained by the very short procedure for which the drugs were used and the relatively small doses in which they were given. Furthermore, sophisticated tests for assessing recovery of neurological function may be necessary to show a significant difference in recovery between these drugs when used in these doses. However, these tests are not universally applicable or useful in the clinical situation.

Midazolam had a much longer duration of action, and it was considered reasonable to reverse its effects with flumazenil 15-30 minutes after the induction of anaesthesia. There was, however, an unacceptably high incidence (50%)

of resedation at the time of interview 4 hours later. This could be explained by the shorter duration of action of flumazenil which has an elimination half-life of 1 (0.2) hours¹⁶ compared to 2 hours for midazolam.¹⁷ The effective duration of action of flumazenil is, however, 1-2 hours¹⁸ while that of midazolam varies from 1.5 to 4 hours, and is dose dependent. Patients who have received flumazenil should be closely observed because of the hazard of resedation.

There was no significant difference in the time to loss of eyelash reflex in the propofol and thiopentone groups. This supports previous findings that the induction characteristics are similar with these two agents.¹⁵ However, there was a significantly longer induction time with midazolam and this too is in accordance with previous findings.

Propofol and midazolam produced a significantly greater decrease in mean blood pressure compared to thiopentone. This persisted during the period under study in the propofol group. Earlier studies with propofol have reported similar results.^{19,20} The mean blood pressure in the propofol group, in contrast to thiopentone, did not return to control values after electrical defibrillation (and consequent sympathetic stimulation). This persistently low blood pressure is an obvious disadvantage in the use of propofol in patients with a compromised myocardium and a low initial blood pressure who need to be cardioverted e.g. ventricular tachycardia. The relatively small decrease in mean blood pressure with thiopentone as compared to midazolam may be explained by the shorter induction time with thiopentone which was followed immediately by cardioversion. This may have resulted in a relatively higher mean blood pressure, while the longer induction time with midazolam caused a continuing decrease in blood pressure. The dose of midazolam in this study was also relatively higher than that reported in two previous studies.^{10,11} There is, however, a large variation in the recommended induction dose for midazolam (0.2-0.4 mg/kg). We felt justified in using a higher dose because the patients were unpremedicated, no analgesics were used during the procedure and because loss of eyelash reflex was used as an end-point to determine anaesthetic depth. In addition, in the small group of four patients studied previously for cardioversion during midazolam anaesthesia,¹⁰ one patient recalled the

procedure. In the second study,¹¹ one of 17 patients was fully aware during the procedure. This may have been because of the relatively small dose in which midazolam was used.

Apnoea after induction with propofol and thiopentone is always a possibility (30%) and facilities for controlled ventilation by an experienced person should be available. In contrast, there were no cases of apnoea following midazolam. A low SpO_2 ($< 95\%$) was more common in the propofol group as compared to thiopentone and tended to occur in the first 10 minutes of anaesthesia. A low SpO_2 was also more common in the midazolam group than thiopentone, but in contrast to propofol, tended to occur after 10 minutes following induction of anaesthesia. This may be explained by the prolonged sleep after cardioversion that occurs in patients given midazolam and possible respiratory depression leading to hypoxia.

Our findings suggest that thiopentone is the drug of choice for cardioversion because of rapid recovery and the least effect on arterial pressure. Propofol has no clear advantage over thiopentone and has the additional problem of a significant decrease in blood pressure. Midazolam, although safe, is clearly not the ideal drug for this procedure.

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Anaesthesia, 1990, Volume 45, pages 875–879

Minitracheotomy: complications and follow-up with fiberoptic tracheoscopy

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Summary

Complications and changes in tracheal mucosa after minitracheotomy were evaluated in 28 patients. Tracheal mucosa was inspected fiberoptically after the insertion of a minitracheotomy cannula, and then at 3-day intervals until the cannula was removed. Thereafter, assessments were made every third day until the mucosa was considered normal. Three significant complications occurred: mediastinal puncture, paratracheal entrance of the cannula and subcutaneous emphysema. Difficulties at insertion of the minitracheotomy cannula were encountered in 15 of 28 patients (54%). Air flow detected through the cannula in one patient, and lack of air flow in another patient, were misleading signs of the position of the cannula. Passing a suction catheter in three patients and a normal end-tidal carbon dioxide tracing in one patient, were also found to be misleading. The

correct position and possible complications could be verified only by fiberoptic tracheoscopy. Changes in the tracheal mucosa were independent of the duration of minitracheotomy therapy.

Key words:

Equipment; minitracheotomy.

Complications; subcutaneous emphysema, misplacement, mediastinal puncture.

Minitracheotomy provides access to the trachea for suction¹ and emergency ventilation.²⁻⁴ Its advantages in treating patients unable to clear secretions from their airway have been demonstrated by Pedersen.⁵ Introduction of the cannula into the trachea through the cricothyroid membrane may be regarded as relatively easy, because the distance from the skin to the tracheal lumen is short and no major vessels or nerves should be injured.⁶ However, a number of complications related to minitracheotomy are reported, e.g. oesophageal perforation,⁷ profuse bleeding⁸ and pneumothorax.⁹ The tracheal mucosa is vulnerable to compression. Mucosal damage after prolonged intubation of the trachea or tracheostomy was reported,^{10,11} but the effect of minitracheotomy on the tracheal mucosa has not been evaluated.

The present study was designed to assess the safety of the percutaneous technique of introducing the cannula into the trachea. The effect of minitracheotomy on the tracheal mucosa was examined by repeated fiberoptic inspection.

Patients and methods

All patients for whom minitracheotomy (MT) was indicated over one year were included in the study (Table 1). MT was performed for treatment of sputum retention in 14 patients and in 14 patients the technique was used prophylactically, immediately after thoracotomy (12) or laparotomy (2). The study was approved by the hospital ethics committee and informed consent was obtained from the patients.

A Mini-trach II set (Portex Ltd., England) was used to insert the minitracheotomy in all cases. The patient's neck was hyperextended and the cricothyroid membrane was identified. Plain lignocaine 1% was used for subcutaneous local anaesthesia and plain lignocaine 4%, 1 to 2 ml, was injected into the trachea. The incision was made with the knife included in the set, but if necessary the incision was deepened with a surgical scalpel number 11. The stilette

was introduced into the trachea and the cannula threaded over it. The correct position of the minitracheotomy tube was verified initially using conventional signs (Table 2); subsequently, fiberoptic examination of the upper airway was performed.

The condition of the tracheal mucosa was assessed fiberoptically immediately after MT and was planned to be performed every third day until the cannula was removed. Fiberoptic tracheoscopy was performed at 3-day intervals after removal of MT until the tracheal mucosa was considered normal or the patient was discharged from hospital, or died. Fiberoptic follow-up was also discontinued if the physical status of the patient was critical. Photographs were taken and a precise drawing of the intratracheal insertion site was made after each assessment. Hyperaemia and swelling at the puncture site were noted. The localisation of the intratracheal entrance was recorded. The tracheal surface was also viewed at the level of the distal end of the cannula for any signs of irritation of the mucosa.

The distal end of the cannula was cultured for microbial growth after removal in 17 patients. Signs of infection at the outer puncture site were noted. Healing of the skin was assessed.

Antibiotics were administered as required by surgery. Any complications during the minitracheostomy therapy were recorded in detail.

ANOVA was used for statistical analysis of parametric data and Chi-squared test with Yates' correction for nonparametric data. The results are presented as mean and standard deviation (SD). Differences were considered statistically significant if *p* was less than 0.05.

Results

Twenty-eight patients were included in the study and 99 fiberoptic examinations were performed. The duration of minitracheotomy therapy was 4.6 (2.5) days when used prophylactically and 11.8 days (7.7) when the tube was

Table 1. Characteristics of the patients and indications for minitracheotomy.

Age; years mean (SD)	M/F	Indications	Number
65 (18)	13/1	<i>Sputum retention</i>	
		Excessive bronchial secretions	11
		Neurological disorder	3
67 (8)	13/1	<i>Prophylaxis</i>	
		Decreased pulmonary reserve	12
		Impaired ability to cough	2

Table 2. Signs of correct position of cannula immediately after performance of minitracheotomy. Number of patients with a positive or negative sign. Number of misleading signs in parentheses.

	Positive	Negative	Total
Air flow through the cannula	26 (1)	2 (1)	28
Passing a suction catheter through the cannula, obtaining secretions	26 (2)	1 (1)	28
Expired CO ₂ tracing	7 (1)	0 (0)	8
Fiberoptic examination of the trachea	27 (0)	0 (0)	27

Table 3. Difficulties related to the performance of minitracheotomy. Number of patients in parentheses. *, complication revealed by fiberoptic examination. †, this patient's data are excluded elsewhere.

Cause of problems	Problems at insertion	Consequences
Soft tracheal wall (2)	Prolongation of procedure (1)	Perforation of posterior wall (1)* MT cancelled (1)†
Short neck (8)	Prolongation of procedure (8) Longer scalpel needed (6)	Stilette paratracheally (8) Bleeding in the trachea (2)* Incision near to vocal cords (1)* Rough edges of incision (1)* Tissue flap in trachea (1)*
Rigid tracheal wall (4)	Prolongation of the procedure (4)	Two cuts in trachea (1)* Tissue flap in trachea (1)*
Skeletal traction (1)	Stilette perpendicularly in trachea (1) Prolongation of the procedure (1)	Cannula paratracheally (1)* None
No apparent cause (2)	Prolongation of the procedure (1)	Incision in the lateral wall of the trachea (1)* Bleeding in the trachea (1)*

inserted to treat sputum retention ($p < 0.01$). Two patients removed their minitracheotomy tube spontaneously, one on the third and the other on the seventh day after insertion. MT treatment was discontinued in two patients because formal tracheostomy was performed and in another two because of tracheal intubation and controlled ventilation. One patient was transferred to another hospital before decannulation. The physiological status of the patient deteriorated in three cases and fiberoptic follow-up was not justified.

Problems with insertion were encountered, and the performance of minitracheotomy was prolonged, in 15 patients (Table 3). The incision was deepened with a scalpel in 10 of 28 patients when the blade of the knife included in the set was too short to reach the tracheal lumen. The changes observed on the intratracheal surface were not related to the instrument used for tracheal perforation.

Fiberoptic examination was performed immediately after insertion of the minitracheotomy. It showed the position of the cannula in all cases (Table 2). The position was incorrect in two patients. In two other cases the tube was guided into the tracheal lumen under visual control during fiberoptic examination. Air flow was felt through the cannula in 26 of 28 patients. In one case, the lack of air flow indicated correctly that the tube was positioned incorrectly, despite the fact that a suction catheter could be passed easily into the mediastinum and a small amount of blood was suctioned. In the other case, the cannula was found to be in the trachea but the lumen was occluded by thick secretions. No air flow was felt and a suction catheter could not be advanced through the cannula into the trachea.

A normal end-tidal CO_2 -tracing was detected in eight patients when the probe (Normocap, Datex, Finland) was

attached to the cannula. The distal end of the MT tube was in the trachea in all eight patients, but in one the tip of the cannula entered the trachea through the posterior wall after traversing the paratracheal tissue.

The changes in tracheal mucosa during and after MT therapy are summarised in Table 4. Hyperaemia and swelling at the puncture site were not related to the duration of MT therapy. Irritation of the posterior wall was seen at the level of the distal end of the cannula in two patients. The mucosa of the posterior wall was normal on the sixth day after removal of the minitracheotomy tube.

Mild local inflammation of the skin was noted in 13 of 18 patients at the time of tube removal. The changes subsided without complications in 3 days and by the sixth day only a small crust was seen at the site of incision. Tracheostomy was indicated in four patients during (two patients) or soon after (two patients) minitracheotomy therapy. Skin healing was not assessed in these patients.

The tip of the cannula was cultured for microbial growth in 17 patients. The results are presented in Table 5. No correlation could be established between microbial growth and the clinical recovery of the tracheal mucosa after cannulation.

A vein was accidentally cut and needed ligation in a patient with a large thyroid gland. In another patient, bleeding from the puncture hole occurred after the patient had returned to the ward. The bleeding was controlled by compression. In one patient the therapy was prolonged (28 days), and the tracheal mucosa appeared infected around the insertion site. The changes subsided in 5 days after removal of the cannula. In one patient, fiberoptic examination showed that the tube had perforated the posterior wall of the trachea, but the oesophagus was intact. A pneumo-mediastinum was seen in the chest X ray after the position

Table 4. Changes in the tracheal mucosa during and after minitracheotomy therapy. T, minitracheotomy inserted for treatment of sputum retention. P, minitracheotomy inserted prophylactically.

	Total number of inspections		Hyperaemia		Swelling		Both changes		No changes	
	T	P	T	P	T	P	T	P	T	P
At insertion	13	14	0	0	0	0	0	0	13	14
3 days after insertion	10	7	3	1	3	2	2	1	6	5
At removal	8	11	4	6	3	5	2	3	3	3
3 days after removal	7	10	3	7	0	6	0	4	4	1
6 days after removal	5	5	4	3	2	1	2	1	1	2

Table 5. Culture of the tip for microbial growth in 17 cannulae. T, MT inserted for treatment of sputum retention. P, MT inserted prophylactically. SD in parentheses.

Microbe	Number of positive cultures	Duration of MT (days)	T/P (number of patients)
<i>Streptococcus β-haemolyticus</i>	1	4	0/1
<i>Streptococcus pneumonia</i>	2	3.5 (3.5)	0/2
<i>Streptococcus faecalis</i>	1	4	1/0
<i>Streptococcus viridans</i>	2	3 (0)	0/2
<i>Serratia marcescens</i>	1	14	1/0
<i>Staphylococcus epidermidis</i>	3	8.3 (6.8)	1/2
<i>Pseudomonas maltophilia</i>	1	7	1/0
<i>Pseudomonas aeruginosa</i>	1	10	1/0
<i>Enterobacter cloacae</i>	1	3	0/1
<i>Branhamella catarrhalis</i>	1	1	0/1
<i>Candida albicans</i>	5	6.0 (3.1)	1/4
No growth	3	4.7 (1.5)	1/2

of the MT had been corrected. In another patient a normal end expired CO₂ trace was obtained and conventional signs of correct position were noted. The cannula traversed paratracheal tissues and entered the trachea through the posterior wall at the level of the second cartilage. Perforation of the oesophagus was not detected.

Subcutaneous emphysema in the neck developed in one patient 20 hours after cannulation. Formal tracheostomy was required to secure the airway. Suctioning irritated the airway in one patient, and rupture of surgical sutures in the abdomen was attributed to strenuous coughing. Two patients complained of discomfort while the cannula was in place.

Discussion

There have only been a few controlled studies of the technique of minitracheotomy. The advantages of minitracheotomy are obvious in the treatment of sputum retention^{1,5} and it has been claimed to be suitable for provision of mechanical ventilation.^{2,4} In recent years, there have been numerous case reports of complications related to minitracheotomy.⁷⁻⁹ Some complications were noted in prospective studies, but only a few were considered severe.^{4,5}

The percutaneous technique of insertion is potentially hazardous due to the blind introduction of the stylette and the minitracheotomy cannula. The stylette of the set used in this study could not always be guided into the trachea through the incision made with the scalpel; this problem can be avoided by using a modification of the Seldinger technique.¹² Furthermore, the stylette could be advanced easily into the paratracheal tissues. Anatomical abnormalities increase the risk for complications.¹³ In our study, problems were encountered most often with patients with a short neck or either too soft or too rigid a tracheal wall.

The position of a minitracheotomy tube must be verified reliably after insertion.¹⁴ In the present study, conventional signs such as passing a suction catheter or detection of air flow through the cannula were misleading in three patients. The correct and safe position of the cannula can be confirmed by fiberoptic tracheoscopy. It is a relatively easy technique and reveals a number of possible complications.

Mucosal changes at the site of tube insertion were seen in 79% of the patients immediately after removal of the MT tube and 3 and 6 days later the frequency was 71% and 70%, respectively. Lomholt *et al.*¹¹ examined the trachea after tracheostomy, and the incidence of mucosal changes varied from 50 to 71% depending on the duration of cannulation. However, the interest was focused on the cuff area rather than the insertion site. In another report,

airway complications after cricothyroidotomy were seen in 52% of patients and were found to be related to the length of cannulation.¹⁵ In the present study, the severity of mucosal lesions was independent of the duration of minitracheotomy therapy.

Tracheal intubation eliminates the nasal bacterial filter and tracheostomies become colonised by bacterial flora.^{16,17} Minitracheotomy causes only minor diversion of breathed air. In our study, bacterial growth was found in 12 MT tubes and *Candida albicans* in five. However, infections that required therapeutic intervention were not found.

Tracheal stenosis has been detected at the level of the stoma in patients with low tracheostomy.¹⁶ The outer diameter of the minitracheotomy tube is small and no compression of the cartilage at the stoma site or of the tracheal mucosa occurs. Thus, the risk for development of tracheal stenosis is probably negligible. However, a long-term follow-up after minitracheotomy therapy has not been reported.

In conclusion, the duration of cannulation after MT does not increase the incidence of complications. Fiberoptic tracheoscopy is a valuable method to verify the correct position of the cannula.

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Failure of nitrous oxide supply to theatre pipeline system

An incident recently occurred that resulted in the interruption of nitrous oxide supply to the theatre pipeline system during an operation. It happened while planned preventive maintenance work was being carried out by a qualified medical gas pipeline engineer.

The anaesthetist concerned was informed when he arrived at the hospital that the engineer would be testing

the gas failure alarms and carrying out routine service and maintenance on the nitrous oxide Ohmeda 301, automatic and standby manifolds. Nothing would be done that would interrupt the gas supply, and the alarm signals should be ignored during testing. The nitrous oxide Rotameter bobbin fell and the pipeline pressure was noted to be zero during the last case on the list. Anaesthesia was continued

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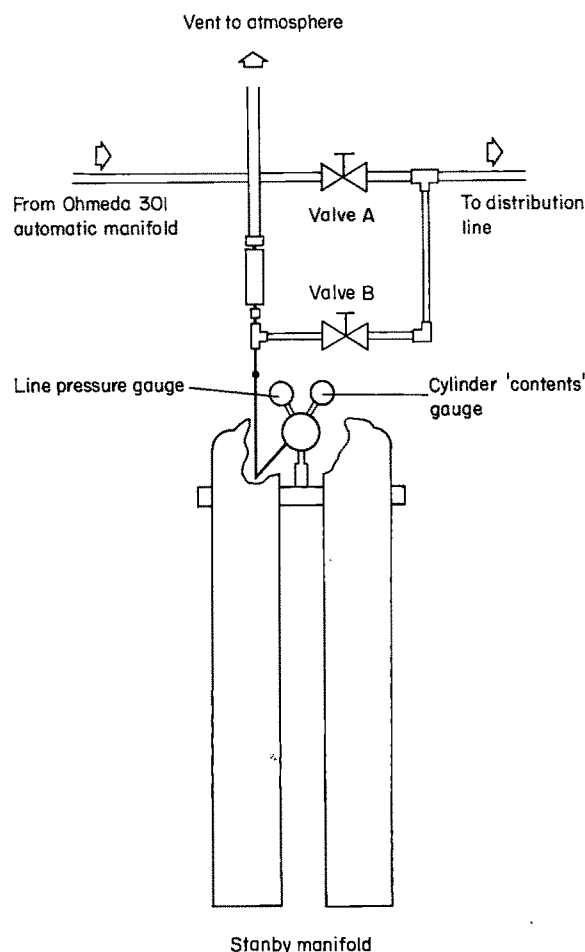


Fig. 1.

using the reserve cylinder on the anaesthetic machine and the anaesthetic was completed uneventfully. The engineer was contacted urgently and informed of the failure and, a few moments later, the pipeline pressure was restored. The sequence of events in the manifold room which led to the failure is described below.

The engineer first checked the standby manifold to ensure the equipment was functioning satisfactorily, so it could be put on line while the automatic manifold was isolated. This allowed the alarms to be tested and the automatic manifold serviced. He confirmed that Valve B (Fig. 1) was closed so that the standby manifold was isolated from the line; the engineer noted that the contents gauge on the standby regulator was reading 20 bar. He opened the right-hand cylinder and heard an increase in pressure and saw that the contents gauge increased to approximately 40 bar. He then opened the left-hand cylinder and found that it was already open, and made the assumption that it was half full since the contents gauge on the first checking was 20 bar. Both cylinders were then closed and the contents gauge observed to ensure that there was no leak, indicated by a decrease in pressure. No leak was detected. The cylinders were opened again and the line pressure gauge checked to ensure that it was reading 4 bar. The output from the regulator was cracked open to ensure that the regulator would maintain 4 bar under flow conditions. This test was satisfactory and the output nut was retightened. Valve B was then opened to bring the standby manifold on line together with the automatic manifold. The right-hand standby manifold cylinder was then closed and at this point both line and content gauges

were reading correct pressures. The automatic manifold was then isolated by closing Valve A to allow tests to be carried out. The anaesthetist received a report shortly after that the nitrous oxide supply had failed. The engineer checked the gauge and noted the contents gauge showed 20 bar but the line gauge showed zero. He immediately turned on the right-hand cylinder and line pressure was restored. Further investigation showed that the left-hand cylinder was empty and that the contents gauge would not decrease below 20 bar, but it would increase when full cylinder pressure was turned on.

The contractors stated, when the incident was discussed with them, that it was common practice throughout the country for engineers to change over from automatic manifold to standby manifolds while the systems were being used to supply medical gases to patients. A 'Permit to Work' was not usually sought under these circumstances because there was no intention to interrupt the gas supply.

General safety rules and procedures for medical gas installations are laid down by the Department of Health,¹ to guard against the sudden disruption of the supply of gases, without the knowledge or permission of medical or nursing staff.

Section 1.1.2 of the Memorandum states: 'Permit to Work Certificates should always be used to control engineering work however minimal, in order to protect patients.' The issuing of a Permit to Work does ensure that alternative arrangements are made to maintain security of gas supplies to patients.

Section 2.3 — Planned Preventive Maintenance (PPM) Work, states: 'Permits will not be required for routine daily or weekly inspections where the service is not interrupted, but follow-up work will usually involve the issue of a Permit. Other PPM inspections will involve the issue of a permit.' It would appear from the contractors' comments that the relevant regulations are open to a less strict interpretation than intended by the Department of Health, and that this practice may be widespread.

This incident raises the whole question of the safety of using a system to supply gases to patients when the protective alarms are disabled. It is essential to ensure that the two cylinders on the standby manifold are kept closed off to make sure that gas does not leak out of the cylinders. The left hand cylinder in this instance had been left on in contravention of the manufacturer's instructions and hospital policy.

Standby manifold cylinders may, of course, stand waiting to be used for many months and presumably, in this case, a slow leak must have occurred at some point between the cylinder connexion to the tail pipe and the faulty pressure gauge.

The porters now rotate the standby manifold bottles onto the main manifold at each routine change of bottles to ensure that full bottles are put onto the standby manifold with reasonable frequency. This ensures that the standby manifold bottles do not stand unused for many months.

The assumption by the engineer that the nitrous oxide cylinder was half full, based on the observation that the contents gauge was reading 20 bar, midway between the maximum pressure registered of 40 bar and zero, was of course erroneous.

The incident described involved a nitrous oxide manifold but the 301 System is also widely used to supply oxygen, in which case failure of the gas supply might have had a much more serious outcome. There are approximately 1000 Ohmeda 301 Units in service throughout the country, mainly in small satellite units.

The incident has been reported to the Department of Health and their comments are awaited. It is important, in view of the serious nature of this incident, to draw

attention to our difficulties so that other Units may review their servicing arrangements.

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Propofol infusions: correlation is not regression; prediction is not achievement

Pharmacokinetic parameters that have been derived from groups of patients cannot be applied to individuals because there is too much inter-patient variability; the parameters are a guide, but no more. White and Kenny (*Anaesthesia* 1990; 45: 204-9) wrote that the measured concentrations of propofol 'correlated closely' with the concentrations predicted by their computer model. It is true that they obtained highly significant correlation coefficients, but the correlation coefficient is a misleading statistic. There could be a highly significant correlation between the heights and weights of 1000 adult men, but it would not be possible to predict the weight of an individual from a knowledge of his height, although one could predict a range of expected weights if given the regression equation and the 95% confidence limits of the observations. So it is with White and Kenny's observations. They give regression equations but, from their Figure 2, these are of limited value for an individual patient: for example, a predicted concentration

of 2 µg/ml resulted in a measured value of between 1.1 and 3.6 µg/ml.

Given that there is such a discrepancy between the actual and predicted concentrations of propofol, it is misleading for them to have written (page 205) that the infusion is altered by the computer program '... when the predicted concentration has been achieved ...', 'until the new predicted value is reached ...', and '... when the new predicted concentration has been achieved ...', because they could not have known what the concentrations were until they had been measured in the laboratory. They meant that the infusion was altered when the computer program predicted that the desired concentration had been achieved, which is not the same as its actual achievement.

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Acute viral myocarditis?

We found the diagnosis of acute viral myocarditis in the case reported by Drs Brampton and Jago unconvincing (*Anaesthesia* 1990; 45: 215-7). Their paper contains fundamental inaccuracies and does not reflect current opinion on acute myocarditis. First, their statement 'microscopic examination of the myocardium ... revealed diagnostic histological signs of acute myocarditis of viral origin' must be challenged.

There are no such 'diagnostic' signs. The histological diagnosis of myocarditis, let alone the acute form, is the subject of considerable debate and controversy.¹ Pathologists round the world use different sets of criteria with chaotic consequences. This is why, for example, the frequency of myocarditis reported from endomyocardial biopsy series has ranged from 3% to 63%.^{2,3} The study by Shanes *et al.* was revealing.⁴ Seven leading cardiac pathologists were shown the same slides and the study came to the conclusion that interobserver variability is high even among experts.

A group of pathologists met in Dallas in 1984 in order to reach consensus and the Dallas classification was formulated; most cardiac pathologists now use this classification to diagnose the various forms of myocarditis. The case report makes no mention whether these criteria were observed. They report focal inflammatory infiltrates, which may not necessarily mean anything,^{1,5} and there is no mention of the extent of these focal areas. Were they numerous and large enough to impair left ventricular contraction? Were the conducting systems involved?

One must also question the validity of the term 'viral' myocarditis. The authors present no evidence whatsoever of viruses being involved. This diagnosis should only be made if one or more of the following can be demonstrated: clinical features of a viral illness, isolation of viruses from

body fluids, rising viral antibody titres and more recently actual viral RNA from the myocardium using virus-specific cDNA hybridisation probes.⁶

We also disagree completely with the statement 'Invariably, there is ST elevation and T wave flattening in all leads'. It is true that the ECG is almost always abnormal in acute myocarditis, but the changes are varied and nonspecific. ST elevation is not common, is seen in less than one third of patients,⁷ and usually signifies myopericarditis.

We do not share Drs Brampton and Jago's optimism in using the electrocardiogram as a screening tool for 'detecting myocarditis'. The ECG is far too nonspecific for this purpose. We do believe, however, that the ECG is part of the pre-operative examination of patients. An abnormal ECG alerts the anaesthetist that the patient may need further evaluation before operation.

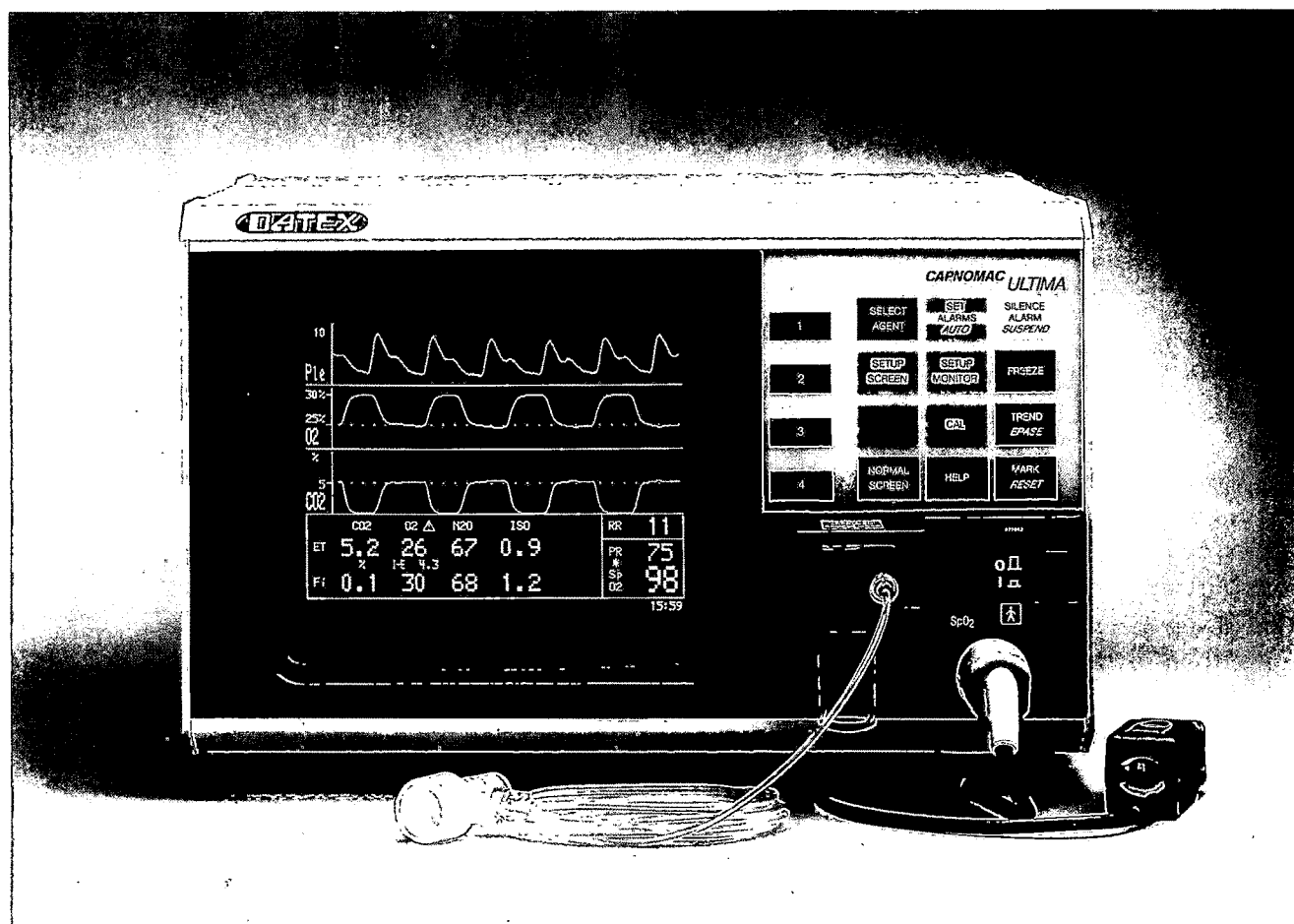
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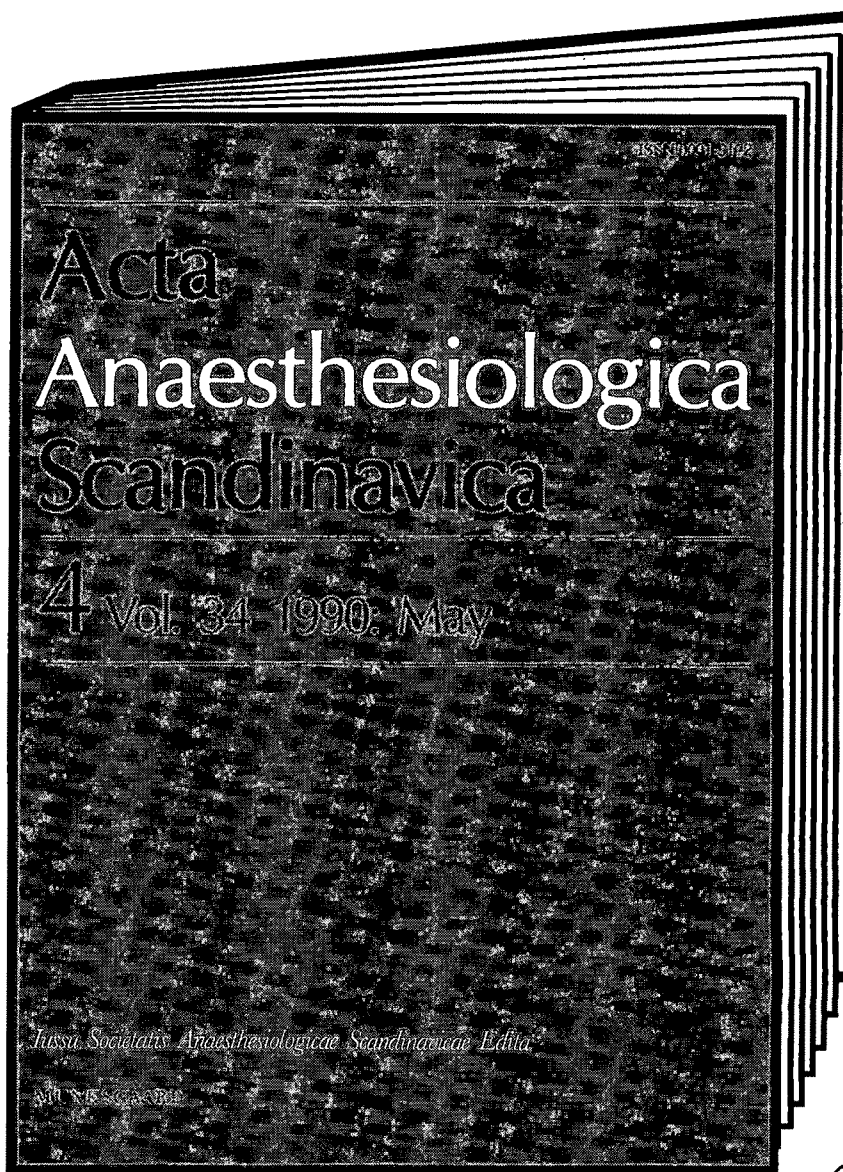
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The death soon after surgery of a young, generally fit patient is a tragedy, but the conclusions reached by Drs Brampton and Jago (*Anaesthesia* 1990; 45: 215-17) are not necessarily valid. They assert that the death was from asymptomatic viral myocarditis and that routine ECG screening before surgery would ensure that this diagnosis was not missed.

The histological diagnosis of myocarditis is not clear cut, and the findings in this case may not actually represent acute viral myocarditis. Focal lesions with inflammatory infiltrates have been shown to develop in previously healthy individuals as a result of stress or the use of vasopressor agents. It is uncertain whether such lesions represent early disease, heralding the fulminant, diffuse changes normally present in severe, acute myocarditis associated with heart failure.¹

There is no evidence to support the statement that routine ECG screening would identify all cases of asymptomatic myocarditis. All patients in one series² were symptomatic and, in 12 of the 22 who had myocarditis without associated pericarditis, the ECG abnormalities amounted only to nonspecific T-wave flattening or inversion, with or without occasional ventricular ectopic beats. It is likely that most anaesthetists would discount such minor changes if the patient appeared fit and asymptomatic.

The reported case teaches us to be more wary and look critically at the ECG of a patient who complains of recent chest pain, pyrexia, or influenza-like symptoms, but I cannot accept that it should bring about the practice of routine pre-operative electrocardiography for all patients.

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A reply

Thank you for the opportunity to comment on the points made by Drs Rittoo and Rittoo and Dr Stewart. Firstly we thank Drs Rittoo for pointing out our error about the ECG changes associated with acute myocarditis. The sentence to which they refer should have read 'Almost invariably there is S-T elevation and (or) T wave flattening or inversion,' but had become corrupted during the drafting process before submission for publication. We cannot, however, accept that there are any other

inaccuracies in the paper that make the diagnosis of acute myocarditis unconvincing.

Our paper was written to describe a clinical episode with a tragic outcome in the hope that our experience would benefit other practising clinical anaesthetists. We deliberately avoided the irrelevant and distracting aspects of myocarditis with which Dr Rittoo and Rittoo's letter is concerned.

The term 'diagnostic' was used to refer to the histological findings in the introduction to the paper for the sake of brevity, and this term is correct in its true sense.¹ Precise details of the histological basis of the diagnosis were given in the final paragraph of the case history and are entirely consistent with the correspondents' assertions about the difficulties of diagnosing this condition. These details were taken verbatim from the report of the independent teaching hospital pathologist who performed the postmortem on behalf of the coroner. The further obsession of the Doctors Rittoo with the histological interpretation of endomyocardial biopsy specimens, as detailed in their references 1-5, are largely irrelevant. In the case reported by us the entire heart and coronary circulation were available for examination, and thus avoided the problems of minute sample size, sampling error and tissue artefact that are associated with endomyocardial biopsy. Inasmuch as the Dallas Classification is relevant, the pathological details we gave show that the definition of myocarditis was fulfilled as 'a process characterized by an inflammatory infiltrate of the myocardium with necrosis and (or) degeneration of adjacent myocytes not typical of ischaemic damage associated with coronary artery disease.'² It should be clear from the context that the viral origin of the disease was a reasonable assumption based upon the clinical history of sudden death in an apparently fit and healthy young woman. We note Dr Stewart's concern about other causes of focal inflammatory myocardial infiltrates, but point out that in this case there was associated myocyte degeneration, as required to fulfil the Dallas definition. Fulminant changes would not be expected since the patient was not in heart failure.

Finally, we agree with all the authors that the ECG is an entirely nonspecific investigation with respect to myocarditis, but represents a sensitive method of excluding the condition. We suggested no more than this in our paper. It is, of course, to be expected that occasional cases of myocarditis will be detected as a result of further evaluation following an abnormal pre-operative ECG recording. There is no published work on the ECG in asymptomatic myocarditis so it is of course impossible to be sure that all cases will be associated with changes. It is, however, the only practical screening tool available for this condition. Careful attention to minor pre-operative ECG changes are important and, as Dr Stewart points out, suggestive signs and symptoms should raise the level of suspicion.

We stand by our case report as presented and believe that the clinical and pathological facts taken together provide a convincing and accurate argument for the extension of pre-operative ECG testing to younger patients who are to be subjected to significant physiological stress.

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Aspiration of hot tea

We read with interest the special article by Mrs D. Brahams (*Anaesthesia* 1989; **44**: 859) about respiratory failure in a young child after aspiration of hot tea. We would like to report our management of a similar case.

A 2-year-old boy was brought by parents to our emergency room because they noticed he had difficulty in breathing after he aspirated some very hot tea from the spout of tea pot at 2230 hours. A general practitioner gave him paracetamol syrup and reassured the family. The parents brought him to the emergency room at 0130 because the baby was becoming more distressed.

He was dyspnoeic, distressed, with whitish material issuing from his nose and mouth; saliva was dribbling out. He had mild stridor, but was not cyanosed.

A white plaque was seen on the palate, and the uvula was swollen on visualisation by spotlight and gentle suction. The lips were oedematous.

He was given hydrocortisone 75 mg intravenously and 30% humidified O₂, and was closely observed. ENT and paediatric consultations were obtained immediately and he was seen by a paediatric registrar and an ENT Senior Registrar who consulted the anaesthetist about the possibility of intubation or tracheostomy.

The baby was taken to the operating room at 0300 hours and consultant anaesthetists attended the case. A size 4.0 mm plain orotracheal (later changed to nasal) tube was passed under general anaesthesia. The epiglottis was enlarged and swollen, but white with a similar plaque in the hypopharynx. The baby was then admitted to ICU, given humidified 30% O₂ via a T-piece, antibiotics and steroids. He was accidentally extubated 12 hours later.

The stridor was then much less, the vital signs were stable and blood gases were adequate. We decided to observe him and continue on the same management. Twenty-four hours later feeding started and the baby was ready to be discharged to the ward.

We think that upper airway obstruction in children should be dealt with by senior specialists in the proper location.

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Pulse oximetry and methylene blue

A spurious reduction in the arterial oxygen saturation as measured by pulse oximetry is well recognised after the intravenous injection of methylene blue.¹⁻³ Methylene blue has an absorption peak of 668 nm, and therefore absorbs most of the 660 nm pulse oximeter light emission. This is interpreted by the pulse oximeter to indicate the presence of reduced haemoglobin, and hence a decrease in oxygen saturation is indicated. We report a case in which a similar fictitious desaturation occurred after the intra-uterine injection of methylene blue, which, to our knowledge, is a new observation.

A fit, 23-year-old woman presented for laparotomy and salpingostomy. A recent diagnostic laparoscopy had shown dense adhesions around the fallopian tubes and ovaries, and injection of methylene blue had revealed a nonpatent right tube. Anaesthesia for this procedure was uneventful.

Premedication was with temazepam 20 mg orally 2 hours before surgery. General anaesthesia was induced with papaveretum 20 mg and thiopentone 300 mg. Vecuronium 7 mg was given to facilitate orotracheal intubation, and the position of the tube verified by auscultation of the chest. Anaesthesia was maintained via a circle system with isoflurane 0.5–1.0% in a 35% oxygen in nitrous oxide mixture. The lungs were ventilated to produce normocarbida, and neuromuscular blockade maintained with intermittent boluses of vecuronium. Patient monitoring comprised: ECG, noninvasive arterial pressure, inspired oxygen, nitrous-oxide and vapour concentrations, end-tidal carbon dioxide concentration, airway pressure and expired minute volume. Arterial oxygen saturation was measured using an Ohmeda Biox 3700 pulse oximeter applied to the left index finger.

The surgeons performed adhesiolysis and excised an ovarian cyst. The oxygen saturation remained between 96 and 98% throughout this. Twenty ml 1% methylene blue was injected, in order to assess tubal patency into the body

of the uterus, after a negative aspiration. The measured saturation decreased within 30 seconds to a low of 72% in the presence of a good plethysmographic waveform. The patient did not become cyanosed, and all other monitored variables remained unchanged. Auscultation of the chest revealed good bilateral breath sounds. The measured saturation returned to 97% after 4 minutes. The subsequent progress of surgery, anaesthesia and recovery was uneventful.

A recent paper highlighted how glycine irrigating solution may be absorbed during transcervical resection of the endometrium, in a manner similar to that which occurs during transurethral resection of the prostate.⁴ Absorption occurs across the exposed endometrial vessels when the intra-uterine pressure is high. A subsequent letter stressed that absorption is greatest when the endometrium is at its most vascular.⁵ Our patient was in the late secretory phase of her menstrual cycle; we suggest that the intra-uterine pressure after the injection of methylene blue may have been sufficient to allow its absorption into the subepithelial capillary plexus, and thus to the systemic circulation.

Routine monitoring with pulse oximetry is an ideal for all patients undergoing anaesthesia. However, the displayed value of oxygen saturation must always be interpreted in the light of the clinical situation.

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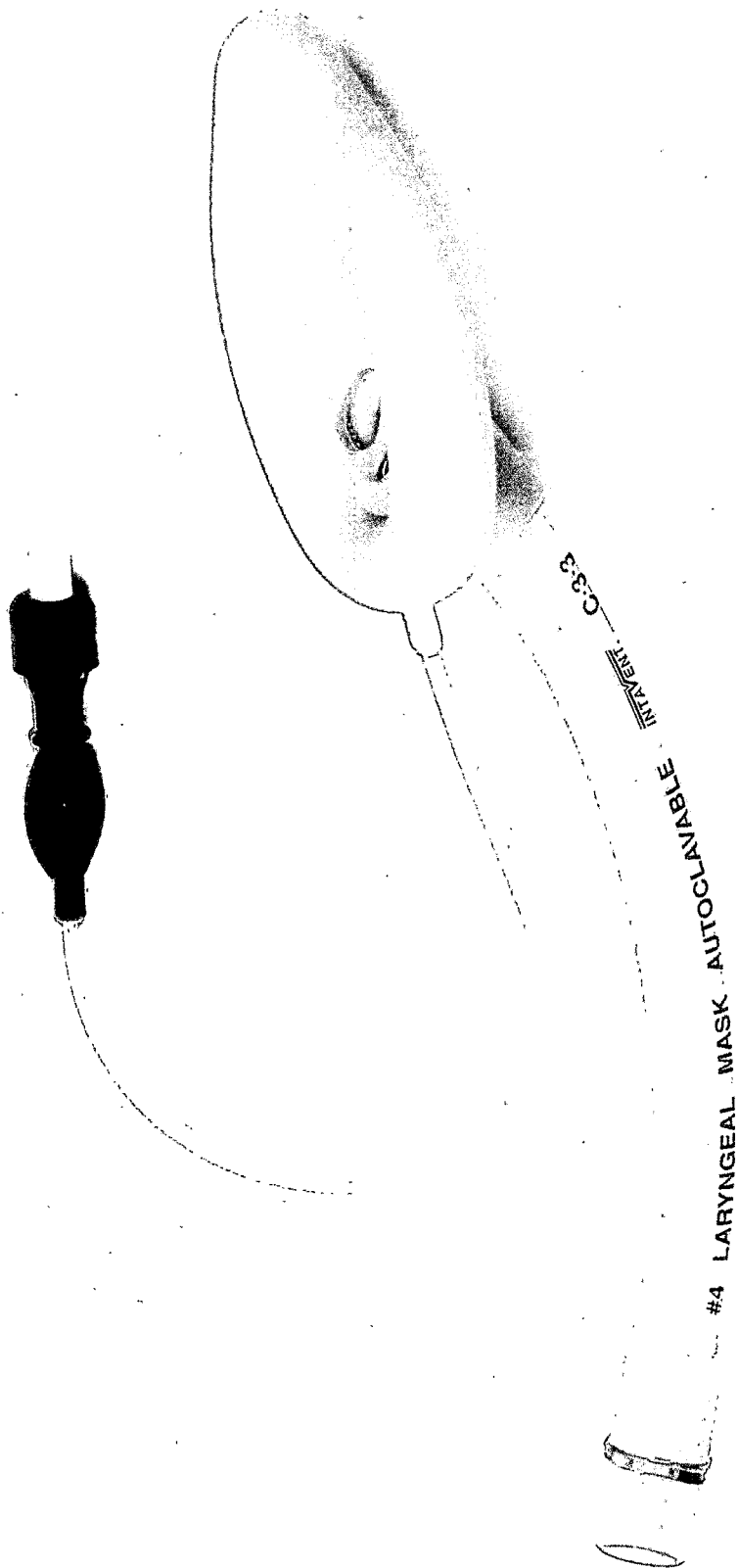
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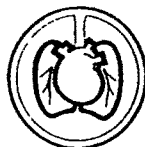


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Dosage or concentration

Dr A.J. Davies (*Anaesthesia* 1990; **45**: 414) states that the use of doses of less than 10 mg bupivacaine has not received much investigation. Reports on the use of such doses are published.¹⁻⁴

1.6 ml 0.5% isobaric bupivacaine has been used for approximately 7 years in about 1000 patients both for Caesarean section and lower limb surgery on elderly patients. Our experience with 0.25% bupivacaine in obstetrics is disappointing.

A randomised prospective trial on 60 patients was started to compare the effects of 1.0 ml 0.75% bupivacaine, 1.5 ml 0.50% bupivacaine and 3.0 ml 0.25% bupivacaine. This was abandoned after inadequate analgesia was produced in five of the first 10 patients in whom 0.25% bupivacaine was used.

The dermatomal spread of the block was similar with all three concentrations but density of block and adequacy of analgesia, while very adequate with both other

concentrations, was often manifestly inadequate with 0.25% bupivacaine.

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Day-case ophthalmic surgery: general or local anaesthesia?

General anaesthesia is commonly used in other surgical specialties for many day-case procedures of up to one hour's duration, so it seems unreasonable not to perform outpatient ophthalmic surgery under general anaesthesia. Current ophthalmic and anaesthetic techniques permit early and safe ambulation in the postoperative period and generally have reduced the necessity for prolonged inpatient management.

Outpatient ophthalmic surgery under both general and local anaesthesia began in this hospital in February 1987. In February 1988 a full-time nurse was employed to coordinate the management of outpatient ophthalmic surgery under both general and local anaesthesia. All patients less than 80 years of age who are not insulin dependent diabetics and are without significant cardiovascular or respiratory disease are considered suitable for outpatient surgery under general anaesthesia. The patients are assessed 2 weeks beforehand by the ophthalmic staff, and an appropriate general medical, ophthalmic, drug and social history (which includes an assessment of the need for district nurse or social services involvement) is taken and a full ophthalmic and medical examination is performed. Investigations are performed (ocular biometry, full blood count and where indicated ECG, serum urea and electrolytes). Patients are instructed in the use of eye drops, and are given written instructions about the day of the operation.

Patients are subsequently admitted fasting for a morning operating list. All patients are required to travel to and from the hospital by private transport and be accompanied by a suitable escort. The medical, social and drug histories are rechecked whilst blood pressure, pulse rate, temperature and weight are recorded; where necessary appropriate eye drops are instilled by the nursing staff.

Both anaesthesia and surgery are performed by either a Consultant or Senior Registrar. The anaesthetic techniques vary according to the nature of the operation and the preference of the anaesthetist. Anaesthesia is induced with either 3-5 mg/kg thiopentone or 1-2 mg/kg propofol and fentanyl 0.5 µg/kg. Neuromuscular block is achieved with vecuronium, and tracheal intubation is performed after topical anaesthesia of the larynx using either 3 ml lignocaine 4% or 3 ml cocaine 5%. Anaesthesia is maintained with nitrous oxide 67% and low concentrations of enflurane or isoflurane in oxygen. Neostigmine 2.5 mg and glycopyrronium 0.5 mg are used to reverse muscle paralysis. ECG, automatic blood pressure, end-tidal carbon dioxide and pulse oximetry are monitored during surgery. Paracetamol 1 g and metoclopramide 10 mg are prescribed for all patients and are administered at the recovery staff's discretion.

The patients are mobilised after 2 hours in the recovery ward. Patients are discharged home with their escort after surgical re-assessment in the early afternoon, in the absence of any anaesthetic complications. They return for surgical review the next day. Patients are followed up one week and one month after surgery and any further complications are recorded at these times.

Five hundred and sixty-six ophthalmic operations were performed as day-case procedures between 1 February 1987 and 31 December 1989. The patients ages ranged from 5 years to 80 years, with a predominance of patients in the age range of 55 to 80 years. One hundred and seventy-six (31%) of the operations were carried out under general anaesthesia. The procedures were extracapsular lens extraction and intra-ocular lens implant, 118; strabismus, 15; other (lid surgery, retinal cryotherapy), 43.

Anaesthetic complications in the group which received

general anaesthesia included one chest infection and one flu-like illness. One patient was re-admitted on the evening of the operation with acute urinary retention. He later underwent a transurethral resection of the prostate. Complications of local anaesthesia during the same period included: 4 retrobulbar haematomata and one doubtful intravascular or intradural injection. This patient required tracheal intubation and ventilation and the surgery was subsequently performed under general anaesthesia. Surgical complications were similar in both groups and included iris prolapse, repositioning of the intra-ocular lens and sterile hypopyon.

There were few complications, despite the age of our patients (up to 80 years) undergoing ophthalmic surgery under general anaesthesia, and no evidence that morbidity was increased by treating them as outpatients. Obviously patient selection is important as is rapid review of any problem that may arise, and admission arranged if necessary. The demand for operations such as cataract extraction will grow as the number of elderly people in the

community increases. Mortality following ophthalmic surgery is extremely low and morbidity, as our study has shown, is not significantly influenced by anaesthetic technique.

We have recently completed a randomised prospective study of cognitive function in elderly patients undergoing cataract extraction under either local or general anaesthesia. This study indicates that at one day and 2 to 3 weeks after surgery there is no significant difference in cognitive function between the two groups. Thus, if anaesthetic facilities are available, selected patients who present for day-case ophthalmic surgery need not be denied, if they or their surgeon so desire, general anaesthesia.

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M. LIM
W. DAVIES
N. CAMPBELL

Postoperative hypoxia

A 6-year-old child was receiving postoperative supplemental oxygen in the recovery room. The anaesthetist was alerted several minutes later, since although the airway was clear and the patient was breathing, he appeared to be cyanosed. The inside of the facemask was misting up, but not demisting with each inspiration. Turning up the oxygen to a higher flow rate caused the oxygen tubing to be detached from the outlet; an obstruction was revealed between the oxygen outlet and the mask. Changing the oxygen mask and tubing rapidly solved the problem.

Closer examination of the mask revealed the oxygen conduit was imperforate (Fig. 1), and that some of the plastic was situated in the mask, which could be an inhalation risk.

The manufacturers (Intersurgical Ltd) inform us that this section of the mask is made by injection of plastic into a

mould. This particular defect was caused by the breaking of the 'core pin' of the mould. Naturally all subsequent masks made from this mould will have the same defect until the fault is remedied.

A sign often used by the recovery room staff is the misting up of an oxygen mask to indicate that the patient is breathing, and the airway is unobstructed. However, if the mask fails to demist during inspiration, obstruction to the oxygen supply should be suspected.

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W.J. FAWCETT
J. ALLT-GRAHAM

A reply

Intersurgical immediately quarantined its stock of these masks on receipt of notification by Dr Allt-Graham. Production records show that a single batch of masks was produced in which a small percentage were faulty. A policy of visual inspection was specified for every mask within this production batch. The failure of this protocol to prevent even one mask from reaching a patient has resulted in a more stringent policy being adopted. The batch would in future be destroyed in order to remove the tedious task of repetitive inspection.

Intersurgical supports Dr Allt-Graham's call to observe as a matter of course the signs indicating oxygen flow to the patient. Intersurgical offers no excuse for this critical fault and has taken action to eliminate its recurrence.

*Intersurgical Ltd,
Crane House,
Gould Road,
Twickenham,
Middx TW2 6RS*

R.B. HICKS

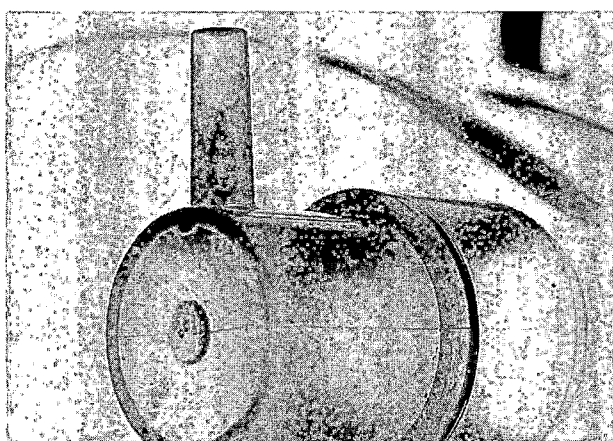


Fig. 1.

Anaesthetic morbidity from trauma to the uvula

Dr Valentine and his colleagues (*Anaesthesia* 1990; 45: 906-8) clearly show a difference in the incidence of sore throat associated with different premedication. They refer to a variety of other causes of this all too common cause of

anaesthetic morbidity but omit to mention an uncommon but recurring cause of sore throat, that is, trauma to the uvula.¹

Trauma to the uvula was found to be the cause of a

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First issue September 1991 Quarterly publication
ISSN 0959-289X

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severe sore throat in a young girl of 18 years of age 3 days after anaesthesia for laparotomy and appendicectomy. She had ceased to complain of abdominal pain by this time. Examination revealed an ulcerated uvula. Suction had been performed using a 'blind' technique and a hard, plastic sucker, before extubation.

Oropharyngeal suction before extubation should be done under direct vision, to avoid trauma to the tissues as well as to confirm that clearance of the secretions is complete. 'Blind' suction is necessary on some occasions if the level of anaesthesia is too light, but sometimes as in this case, it is performed when suction under direct vision is possible. Damage to the uvula is most likely to occur with the hard, plastic suckers which may suck the uvula firmly into their tip. The metal Yankauer sucker with its rounded protective cap is not likely to do this, and is preferable.

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A reply

We thank Dr Stubbing for his comments and note with interest his report of trauma to the uvula as a further cause of postoperative sore throat.

All the patients in our study had oropharyngeal suction under direct vision, using a hard, plastic sucker. None had evidence of uvula trauma at the postoperative interview and examination of their oropharynx. The metal Yankauer design with rounded end may indeed be less damaging to the tissues, but our experience suggests that the plastic sucker with the possible advantage of disposability is adequate if used with appropriate care.

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The interpretation of results by doctor technicians

Verma, Dhond and Lawler (*Anaesthesia* 1990; **45**: 412) raise some important points about the use of laboratory machines by clinicians, and some of the problems which may arise from this. It is appropriate that they should highlight measurement of plasma ionized calcium concentration (cCa^{2+}), since problems are particularly likely to arise in the case of this ion.

Unlike other commonly measured ions, the calcium ion in plasma exists in equilibrium with calcium in two other phases, protein-bound and in combination with anions.¹ This equilibrium may be upset by any factor which influences chemical equilibria, such as pH, temperature or addition of other calcium-complexing substances (including heparin). Plasma cCa^{2+} is therefore particularly likely to be affected by errors in sample collection, storage and handling.

The effect of heparin is predictable and dose-dependent. Concentrations of heparin below 10 IU/ml do not affect plasma cCa^{2+} whereas higher concentrations progressively reduce cCa^{2+} .² Sample heparinisation by filling the syringe deadspace with intravenous sodium heparin 1000 ml, a common practice in intensive care units, gives a concentration of heparin sufficient to reduce cCa^{2+} .³ It is the concentration of heparin, not the form of heparin used, which affects cCa^{2+} . It is likely that the authors' sodium-heparin group yielded lower cCa^{2+} than the lithium-heparin group simply because there was more heparin present.

The use of a clotted sample as a standard for cCa^{2+} is questionable, since the process of clotting has unknown effects on cCa^{2+} . In fact, the authors' results show rather low cCa^{2+} even in the clotted samples: mean $\text{cCa}^{2+} = 1.13$ mmol/litre compared with the usual normal range of about 1.15-1.25 mmol/litre.⁴ Unfortunately the particular electrode system used is not stated, nor is the normal range for that system.

Plasma cCa^{2+} is measured most accurately and conveniently by using a measured concentration of heparin, avoiding contact of the sample with air to prevent pH changes, and performing the measurement immediately since sample storage may have unpredictable effects on cCa^{2+} .⁴

The attraction of having various aspects of blood chemistry immediately measurable in the ITU is undoubted, but Verma *et al.* are correct to draw attention to the limitations of such systems. However simple and accurate the machines are, the answers are meaningless if samples are collected and handled inappropriately. Measurement of cCa^{2+} is particularly fraught with problems, and so requires a different sampling technique; it is debatable whether calcium electrodes should be included as a 'bonus' in machines designed primarily for the measurement of other variables.

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M.P.D. HEINING

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The requirement of intensive care support for the pregnant population

The paper by Drs S.G. Graham and M.C. Luxton (*Anaesthesia* 1989; **44**: 581-5) prompted us to investigate the figures for a nonteaching hospital obstetric unit.

We have a 50-bedded obstetric unit located 220 metres from the main hospital; the latter contains a six-bedded intensive care unit (ICU). All critically ill obstetric patients and a variable number of high dependency patients are admitted to the ICU. We analysed all those patients who required admission to the ICU between 1 January 1985 and 31 December 1989 using the same criteria as Graham and Luxton.

There were 9425 deliveries; of which 13 were admitted to the ICU. Haemorrhage was the most common cause for admission (30.8%); three postpartum and one antepartum. Anaesthesia accounted for 23% ($n = 3$); there were two cases of aspiration and one suxamethonium apnoea confirmed by laboratory tests. Asthma was precipitated during anaesthesia in three cases (23%). Two cases of severe pre-eclampsia (15.4%) and a case of supraventricular tachycardia at Caesarean section (7.7%) completed the list. There were no deaths during the period of the survey.

Seven of the 13 patients (53.9%) remained on the ICU for less than 48 hours; the mean duration of stay was 3.5 days. Six patients required ventilatory support. The mean duration of mechanical ventilation was 2 days.

Six (46.2%) had indwelling intra-arterial cannulae; five (38.5%) had central lines with no pulmonary artery catheters used. Inotropic support was never used, but in the last 2 years, all critically ill patients were given prophylactic renal dose infusions of dopamine.

Seven of the 13 patients (53.8%) had no coagulopathy. However three (23.1%) needed more than four units of blood. The first was a known case of Von Willebrand's disease; the second needed 18 units of blood to keep up with the severe haemolysis as a result of the HELLP syndrome (Haemolysis Elevated Liver and Low Platelets).¹ The third case was a PPH at 34 weeks after vaginal delivery of a dead fetus. She developed disseminated intravascular

coagulation initially, followed by a severe pulmonary embolus 3 days later.

Hypertensive diseases in pregnancy accounted for two cases; one, a severe pre-eclampsia superimposed on essential hypertension, and the other the case of HELLP. Control of hypertension proved difficult in both; both were discharged on oral antihypertensives and at subsequent post-natal checks remained normotensive.

Our retrospective study did not reveal any cases of adult respiratory distress syndrome. However, there were three cases of asthma precipitated at the time of induction of general anaesthesia for emergency section. One of these was a 17-year-old primigravida whose previous asthma history had not been elicited. She was also a heavy smoker. She developed status asthmaticus at induction, received controlled ventilation of the lungs for 9 days in ICU and made slow recovery. She subsequently gave up smoking.

Nine of the 13 patients (69.2%) made full recovery, four (30.8%) had some degree of slight or moderate disability.

The true figures for the life-threatening complications are difficult to establish, it is even more difficult to ascertain the near misses.

Our data reveal that there were 1.38 admissions per 1000 deliveries. However, if there was a distinction made between high dependency and intensive care patients (the latter needed ventilation and (or) intensive monitoring) then only nine were ICU cases. This gives a 'corrected' figure of 0.96 admissions per 1000 deliveries; compared to Graham and Luxton's figures of 1 per 1000!

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APACHE II expected outcome calculations are subject to inter-observer variation

A statistical evaluation of expected outcome of patients in Intensive Therapy Units (ITU) can be made using the 24-hour worst APACHE II score and the one principal reason for ITU admission.¹

The authors independently derived expected outcomes for 10 consecutive ITU admissions using the method described by Knaus *et al.*¹ using a personal computer. The patients were known to all the authors and included medical and postoperative surgical patients whose APACHE II scores ranged from 15 to 30.

APACHE II scores, independently calculated, were not subject to disagreement. The authors made their own assessments of the principal reasons for ITU admission. All four authors agreed on the outcome score in only four of the 10 patients, and agreement occurred between three authors in one patient and between two authors in four patients. There was total disagreement in the remaining

patient with the estimated risk of hospital death ranging from 18 to 56%.

A problem of interobserver variation in calculated expected outcomes of ITU patients is apparent. The APACHE II score is an objective assessment, with interobserver agreement quoted at 96% for the acute physiology score;¹ but the calculation of expected outcome is very dependent on the principal reason for ICU admission. This appears to be subject to differences of opinion.

We suggest these simple observations should be taken into account whenever expected outcome figures, calculated by more than one observer, are compared between, and within, ITUs.

We recommend that while junior staff, who rotate through ITUs, may calculate APACHE II scores, expected outcomes should be calculated by a single permanent

member of staff, as occurs in this ITU. This will allow valid, and meaningful, within-unit comparison of outcome figures. The problem of between-unit comparison remains, particularly when the principal reason for admission is in doubt, e.g. multiple system organ failure.

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Vecuronium and sepsis

Vecuronium is thought to have the fewest direct cardiovascular effects and to have a low tendency to cause the direct release of histamine. We report an episode of hypotension after the administration of vecuronium to a septic patient.

A 86-year-old woman who weighed about 40 kg had an episode of nontransmural myocardial infarction 4 years ago, and since then nifedipine was given to treat her hypertension. Intestinal obstruction from sigmoid colon cancer was diagnosed. An emergency operation was planned after 5 days. She had a history of urticaria caused by some sort of antipyretics. Eosinophils comprised 0.1% of the total white blood cells (12 900 cells/mm). She was drowsy on arrival in the operating room; her arterial blood pressure was 155/65 mmHg and her heart rate was 55/minute. Her respiratory rate was 40/minute; rectal temperature was 35.5°C. Her acid base status was as follows: pH 7.382, P_{aCO_2} 2.0 kPa, HCO_3^- 9.3 mmol/litre, base excess -12 mmol/litre. The blood lactate level was 97.5 mg/dl. A prophylactic intravenous infusion of nitroglycerin was started at a rate of 0.1–0.3 (μ g/kg)/minute. Anaesthesia was induced with thiamylbarbitone 10 mg, fentanyl 0.1 mg and vecuronium 4 mg. The blood pressure decreased from 165/72 to 95/50 mmHg within 10 minutes. The heart rate decreased from 60 to 55 beats/minute. The trachea was then intubated without any increase in blood pressure and heart rate. Intravenous infusion of dopamine at a rate of 1–2 (μ g/kg)/minute was started to maintain visceral blood flow. Her blood pressure gradually increased to 140/60 mmHg. The sigmoid colon was perforated and the peritoneal cavity was completely soiled. Vecuronium 4 mg was administered again, 80 minutes after the initial injection. The blood pressure decreased from 140/60 to 95/50 mmHg after the injection without any change in the heart rate. The blood pressure then increased gradually and reached 120/50 mmHg within 13 minutes.

The first episode of hypotension may not have been solely as a result of the vecuronium administration, but it is most likely that vecuronium was responsible for the second. The possibility of an accidental flush of nitroglycerin is ruled out, because nitroglycerin and

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vecuronium were given through separate intravenous lines. There was no skin manifestation and the blood histamine level was not measured, but it is possible that histamine was involved in this or these episode(s).

Some reports of histamine release after vecuronium administration have appeared.^{1–3} It was also shown that blood histamine levels increase during sepsis.⁴ The patient had, furthermore, a past history of drug allergy. It is possible to speculate that a small increase in histamine level by vecuronium administration is critical for those patients whose blood histamine level has already increased, or that histamine is released more easily by vecuronium in septic patients than it is in normal subjects. It also seems possible that the reactivity to histamine is increased during sepsis.⁵ The simultaneous administration of nitroglycerin might have affected the cardiovascular effect of vecuronium in our patient.

There is a possibility that vecuronium administration induces hypotension in septic patients, and therefore should be used carefully with septic or critically ill patients.

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Propofol for patients with Huntington's chorea?

Huntington's chorea is a rare disease of the nervous system with an atrophy of the basal ganglia that mainly affects the caudate nucleus. The main symptoms include chorea, ataxia, and dysarthria. Anaesthetic experience is limited;

reported problems include prolonged effect of thiopentone¹ and suxamethonium,² generalised tonic spasms induced by postoperative shivering,³ increased sensitivity to midazolam³ and nondepolarising muscle relaxants,⁴ and

increased risk of pulmonary aspiration.⁴ The use of propofol for induction and maintenance of anaesthesia seems to eliminate many of the above problems.

A 42-year-old female with a 6-year history of Huntington's chorea, adipositas permagna (108 kg, 164 cm), and blindness (retinitis pigmentosa) presented for elective repair of an acromioclavicular luxation. Choreatic symptoms had improved when treated with haloperidol 2 mg three times a day, alprazolam 2 mg three times a day, and tiaprid 200 mg three times a day, while a certain dysarthria and loss of intelligence were detectable. Communication was slowed but possible. Past medical history and laboratory results were otherwise unremarkable. A difficult tracheal intubation was anticipated.

Anaesthesia was induced with intravenous propofol 120 mg after pre-oxygenation. Manual ventilation of the lungs by mask was possible, and relaxation was, therefore, accomplished with intravenous atracurium 50 mg. Tracheal intubation was performed after intravenous fentanyl 0.1 mg and confirmation of complete relaxation (train-of-four). Anaesthesia was maintained with an infusion of decreasing amounts of propofol: 10 (mg/kg)/hour for the first 10 minutes, followed by 8 (mg/kg)/hour for another 10 minutes, and then 6 (mg/kg)/hour until the operation was finished. The lungs were ventilated with an air-oxygen mixture and the minute volume was adjusted according to the end-tidal CO₂. Intravenous atracurium (10 mg) and intravenous fentanyl (0.05 mg) had to be repeated only once with nearly complete twitch recovery after 40 minutes and stable haemodynamics within 20% baseline. A sudden decrease in oxygen saturation together with a loss of breath sounds over the right lower lobe after 50 minutes were caused by mucous obstruction and treated with tracheal suction. The patient opened her eyes on command 7 minutes after termination of the propofol infusion and after another 4 minutes she was able to tell us her birth date. Recovery was completely uneventful; no shivering or choreatic movements were observed and the patient felt well.

A preferred anaesthetic technique has not yet been described for patients with Huntington's chorea. The uncertain effect, associated with the duration of thiopentone can be circumvented by using alternative induction agents. Diminished cholinesterase activity as a reason for the prolonged effect of suxamethonium could not be substantiated by other authors.⁵ Our patient had, as measured postoperatively, normal cholinesterase values, and also showed no increased sensitivity to atracurium. Avoidance of inhalational agents and maintenance of normothermia greatly reduces the risk of postoperative shivering. This can be accomplished using a totally intravenous anaesthesia technique. The patient must regain airway protective reflexes as early as possible after anaesthesia to minimise the risk of postoperative pulmonary aspiration. Propofol proved to be effective. Furthermore, the triggering effects which could cause a choreatic crisis were not observed. A delay in recovery compared to a normal patient population was not found.

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Unpleasant sequelae of benzodiazepine sedation

The letter by Dundee (*Anaesthesia* 1990; **45**: 336) on unpleasant sequelae of benzodiazepine sedation in adults makes interesting reading. Recently we completed a study on pre-anaesthetic medication with rectal midazolam in children. Our study¹ showed that midazolam dosage was strongly associated with the prevalence of side effects (Table 1). A distribution of observed side effects are shown in Table 2. Agitated excitement and restlessness were most frequently observed.

We would like to call the unpleasant sequelae that we found paradoxical reactions. But what is a paradoxical reaction? 'Behavioural toxicity is a phrase used to denote those pharmacological reactions of a drug that, when administered within the dosage range in which it has been found to possess clinical utility produce, through mechanisms not immediately specifiable, alterations in perceptual and cognitive functions, psychomotor performance, motivation, mood, interpersonal

Table 1. Contingency table for side effects after drug administrations.

Prevalence of side effects	Experimental group				Total
	Midazolam 0.25 mg/kg	Midazolam 0.35 mg/kg	Midazolam 0.45 mg/kg	Saline control	
None	20	16	10	20	66
	100.0	80.0	50.0	100.0	82.5
Yes	0	4	10	0	14
	0	20.0	50.0	0	17.5
Total	20	20	20	20	80
	25.0	25.0	25.0	25.0	100.0

Chi-squared 23.2035; df 3; $p < 0.0001$.

Table 2 Observed adverse reactions in experimental groups.

Adverse reaction	Midazolam 0.35 mg/kg (n = 4)*	Midazolam 0.45 mg/kg (n = 10)*
Agitated excitement	4	4
Restless/irritated	2	8
Uncooperativeness		2
Disorientation/confusion		5
Emotional/crying	1	3
Visual disturbances		2

* Number of individuals with adverse reactions.

relationships or intrapsychic process of an individual to the degree that they interfere with, or limit the capacity of the individual to function within his setting or constitute a hazard to his physical well-being'.²

Paradoxical reactions characterised by agitated excitement, mental confusion and uncooperativeness are reported after intravenous midazolam.³

We have done extensive studies on the use of intravenous midazolam,⁴ sublingual lorazepam and intramuscular diazepam⁵ for sedation during dental procedures. No signs of paradoxical reactions were found in these studies.

We are left with two impressions on review of the literature. First, the overall incidence of significant paradoxical reactions to rectal midazolam is extremely small, and that few controlled studies exist which define the population at risk for these reactions.

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Opioid supplementation during propofol anaesthesia

We read with interest the article of Moffat *et al.* (*Anaesthesia* 1989; **44**: 644-7). We studied a similar induction technique and found a much lower dose of propofol to be effective in most of our patients.

Thirty three patients ASA class 1 to 2 aged 17 to 70 years who presented for minor gynaecological procedures were studied. No premedication was given. Intravenous fentanyl 1 µg/kg was given 3 minutes before induction with propofol 1.5 mg/kg. Supplementary doses of propofol were given to a maximum of 2.5 mg/kg for induction. Anaesthesia was maintained with nitrous oxide and halothane in oxygen.

Induction was successful in 32 patients (97%) with propofol 1.5 mg/kg. The mean induction dose reported by Moffat *et al.* was 2.67 mg/kg, whereas Thomas *et al.* used fentanyl 100 µg before induction and reported a mean induction dose of 1.98 mg/kg.^{1,2} Our study suggests a much lower dose of propofol is required in Malaysian patients.

The average decrease in mean arterial pressure 3 minutes after induction was 20.7 mmHg (SD 18.3) while the mean decrease in heart rate was 11.4 beats per minute (SD 10.7). The extent of cardiovascular depression observed appears to be greater than that reported by both Moffat *et al.* and Thomas *et al.*, although we used a lower dose of propofol.

Severe cardiovascular side effects are reported after

combined use of propofol and fentanyl.³ We attempted to evaluate the safety of 2 mg/kg propofol as a bolus dose over 60 seconds. Marked hypotension which required treatment with intravenous fluids or ephedrine occurred in two out of eight patients. We subsequently decided to restrict the initial dose to 1.5 mg/kg.

Propofol is currently the standard induction agent for short procedures. Our results, however, do suggest that there may be variation in response between different population groups.

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Will we ever learn?

Your readers must be told of a possible hazard attendant on the use of the Brain laryngeal mask, although I admit my own role in the story rather proves the adage that it is impossible to make things foolproof, because fools are so ingenious.

The patient was a 53-year-old female for excision of a breast lump. She had a rather underdeveloped mandible, so to avoid the toil of holding on a facemask, a number 3 laryngeal mask was used after an induction of anaesthesia with 15 ml profolol (with lignocaine) and 50 µg fentanyl.

The patient remained apnoeic, but the lungs were easy to ventilate by hand. Assuming that spontaneous ventilation would soon resume, I was quite happy to leave things so, since monitoring included both capnograph and pulse oximeter. For some reason now beyond me, I did not inflate the cuff: occasional manual inflations produced satisfactory ventilation.

Unbeknown to me, my anaesthetic nurse *had* inflated the cuff. When the tone of the patient's respiratory muscles returned, my hand-inflation produced a gurgle in the pharynx so I put another 20 ml of air into the cuff. Breathing became absolutely obstructed at once. Immediate removal of the apparatus (after removal of the air from the cuff) produced a clear airway, and anaesthesia was continued with first a facemask and later, for convenience, with a tracheal tube. It was noted during the passage of the latter that the patient had a very large and floppy epiglottis.

The mask was still inflated when it was removed. The effect of the extra 20 ml was to overstretch the cuff and to produce a bulge. This bulge was presumably the cause of the airway obstruction perhaps by pressure on the large epiglottis.

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A reply

Thank you for the opportunity to reply to these comments.

It should be noted that significant overinflation of the cuff of the laryngeal mask can cause herniation and it is therefore important not to exceed the volumes recommended in our literature. It is also emphasised that the cuff should be tested before use and discarded if it inflates unevenly. This may indicate excessive stretching as a result of autoclaving with air in the cuff or accidental overinflation during previous use.

Further information on the laryngeal mask can be obtained from our Instruction Manual; copies are available from the undersigned.

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Partial obstruction of the laryngeal mask airway

We are assessing the position of the LMA with a fiberoptic laryngoscope in anaesthetised children who were continuously monitored by pulse oximetry and were interested to read the recent correspondence (*Anaesthesia* 1990; **45**: 487-8) concerning kinking of the laryngeal mask airway (LMA). From our experience of 90 cases (including two kinked size 2 LMAs) partial airway obstruction involving the LMA is not easily detected by clinical observation: asynchronous respiratory movements of the patient and anaesthetic reservoir bag, indrawing of intercostal spaces or supraclavicular fossae and presence of extraneous airway sounds. Additionally, pulse oximetry in patients who are breathing an oxygen enriched anaesthetic mixture does not provide an early indication of airway obstruction.

Fiberoptic bronchoscopy indicates that in adults about

10% of masks are associated with partial airway obstruction.¹ Our own (unpublished) observations with the fiberoptic laryngoscope show that this figure is higher in children and is between 25 and 50%. We endorse the authors comments about the need to avoid complacency.

The CAT scan picture provided by Goldberg *et al.* seems to show a taut pilot tube. It may be possible that this contributed to kinking the LMA in this instance.

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A modified Intavent laryngeal mask for ENT and dental anaesthesia

The Intavent laryngeal mask (LM) has been widely used in this Health District as a general purpose airway for elective surgery since its introduction 2 years ago. However, its use in ENT and dental anaesthesia is limited by the compressibility and the wide bore of the tubing. We have recently had the opportunity to carry out an informal assessment of prototypes fitted with smaller diameter flexometallic tubes (Fig. 1).

The modified LM consists of a standard, size 3 or 4, mask sealed to an armoured narrow-bore tube of 10-mm internal diameter and 19-cm length. The tubular

components of these LM's fit snugly between the jaw of the split tonsillar gag and allow a clear airway to be secured and maintained during surgery. Insertion and correct placement of this type of LM is more difficult to achieve due to the floppiness of the tube. However, grasping the LM at the junction of the cuff and the tube and adhering to the recommended insertion technique resulted in successful placement in all 20 tonsillectomies. Our ENT colleagues found it possible to insert and fully open the tonsillar gag with the LM in place and strapped down to the centre of the chin.

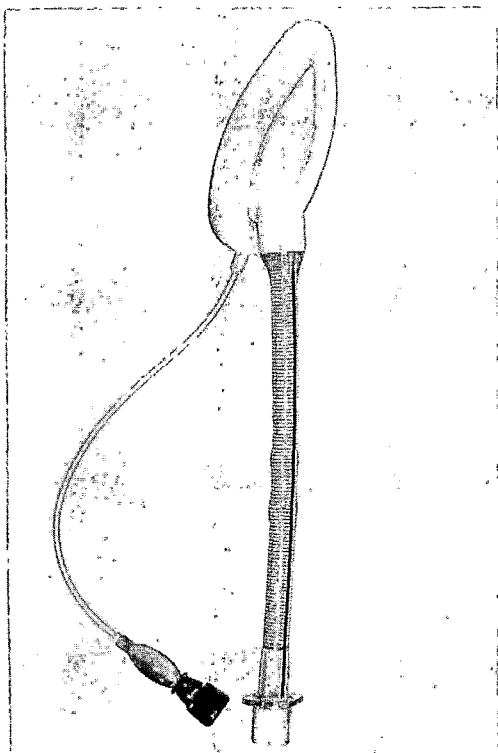


Fig. 1.

There was neither significant airway obstruction nor loss of seal in paralysed or spontaneously breathing patients amongst the 20 for tonsillectomy. It was, however,

necessary to check the patency of the airway when the gag was extended and before surgery was commenced. Access for tonsillar surgery was satisfactory. The gag was removed, the patient placed in the lateral position on completion of surgery and, whilst still anaesthetised, allowed to recover consciousness with the LM in place and inflated. The LM was deflated and removed in the normal manner when the patient awoke and regained protective reflexes. The absence of significant coughing or any stridor at any stage of the procedure indicated good protection of the larynx from blood seepage.

Dental anaesthesia, with spontaneous ventilation for extraction of a range of teeth, (including rear molars) was provided on 10 occasions. The three dental surgeons reported satisfactory access for intra-oral surgery. It was necessary to secure the tube with cotton tape at the mouth rather than use adhesive strapping since this permitted lateral movement of the tube by the surgeon. Movement from one side of the mouth to the other was tolerated without loss of cuff seal as a result of the flexibility of the tube. It was not necessary to insert a pack into the hypopharynx as a routine because the dental surgeon's pack and the seal of the inflated mask adequately contained any secretions and blood. There may be loss of a good LM seal if insertion of a deep pack is attempted.

The great advantages of this technique for dental and tonsillectomy anaesthesia are the avoidance of suxamethonium, the absence of nasal trauma from intubation, and the smoothness and safety of the recovery.

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A supraglottic oropharyngeal airway

In 1983 Brain described the laryngeal mask which is a new concept in airway management.¹ More recently Boheimer *et al.* described their early experience using an alternative device known as a self-retaining nasopharyngeal airway, which is effective during spontaneous breathing.² There is a similar device which is used through the mouth. It consists of a 9.0-mm internal diameter cuffed oral tube, which is shortened to a length of 11 cm. The patient end of the tube is rounded, so that it is without a bevel. Two guide marks are placed around the circumference of the tube at its

machine end, one at 9 cm and the other 11 cm from the tip (Fig. 1). A 15-mm standard plastic connector was inserted into the machine-end of the tube to the level of the 9-cm mark. A specially constructed device has now been produced by Portex with a profile cuff.

The prototypes of this device were used to maintain airway in 100 anaesthetised, adult patients during spontaneous ventilation. Anaesthesia was induced in all patients after diazepam premedication with either thiopentone 4.5 mg/kg or propofol 2–2.5 mg/kg.

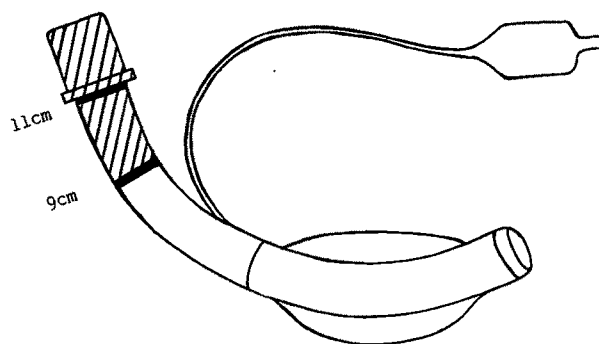


Fig. 1. A supraglottic oropharyngeal airway with two guide marks placed around the circumference of the tube at 9 cm and 11 cm from its tip.

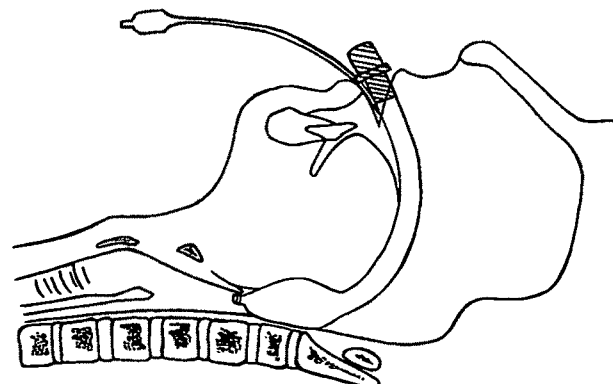


Fig. 2. A supraglottic oropharyngeal airway with its tip resting behind the apex of the epiglottis.

Maintenance was with nitrous oxide, oxygen and isoflurane with the patient breathing spontaneously. When surgical anaesthesia was attained a lubricated tube was placed orally under direct vision using a laryngoscope with the tip of the tube resting behind the apex of the epiglottis (Fig. 2). Successful placement was indicated by conduction of breath sounds up the tube. The cuff was then inflated with 15–25 ml of air to displace the tongue from the posterior pharyngeal wall and secured in place by adhesive tape. The patency of the airway was confirmed by observation of movement of the reservoir bag, capnography and pulse oximetry. The tube was left in place at the end of surgery until the patient recovered consciousness in the recovery ward.

The quality of the airway was assessed as obstructed, partially obstructed, or clear and any complications noted. The airway was used successfully in 94% of patients and a clear airway was maintained. In six patients there was partial obstruction and in four patients partial obstruction was relieved by lifting the jaw. Two patients developed laryngospasm possibly caused by light anaesthesia. This led to partial obstruction which was relieved by deepening the level of anaesthesia. None of the patients needed tracheal intubation.

The laryngeal mask airway and facial burns

The pain experienced by burned patients during dressing changes may be so severe that general anaesthesia may be the only way of achieving freedom from pain. Access to the burnt areas in patients with facial burns often require tracheal intubation which may be technically difficult since suxamethonium is often contraindicated. We have therefore used the laryngeal mask airway as part of a general anaesthetic technique for patients with severe facial burns. Our experience to date has confirmed that it is a useful adjunct for airway control in patients with facial burns, and has so far produced no complications directly attributable to the device.

The distance from the tip of the tube to the incisor teeth was measured in each case; the average distance was 10 cm (SD 1.0). This simple device, which is relatively inexpensive and disposable, seems to function effectively to maintain a clear airway during spontaneous breathing. The cuff not only displaces the tongue from the posterior pharyngeal wall and helps to maintain a patent airway, but also seems to produce an adequate seal in the pharynx so that the anaesthetic gases are not diluted by entrained air. It was also possible to assist ventilation when necessary. A further evaluation is now in progress to determine whether this device can also be inserted blindly.

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There is no need for tracheal intubation and yet excessive handling of the facial burns by the anaesthetist is prevented. This is aesthetically more desirable for the anaesthetist and it minimises the possibility of delayed healing and infection. We believe that the laryngeal mask airway represents a significant advance for burned patients for whom general anaesthesia is necessary when dressing changes are required in such patients every 24 or 48 hours.

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The use of a laryngeal mask airway for CT radiotherapy planning and daily radiotherapy

We describe the use of a laryngeal mask airway (LMA) during anaesthesia for computerised tomographic (CT) radiotherapy planning and daily radiotherapy in a child.

It is our usual practice, when a child requires an anaesthetic for cranial irradiation to use a combination of intravenous ketamine and diazepam via a Hickman catheter after oral atropine.¹ The incidence of serious emergence phenomena with obvious distress was virtually zero in 482 general anaesthetics.

We recently managed a 4-year-old with an intracranial rhabdomyosarcoma who needed cranial irradiation and adjuvant chemotherapy. This child became so disturbed by emergence phenomena after ketamine and Diazemuls, given for the making of a face mould, that a different technique was thought advisable for his subsequent CT radiotherapy planning.

An LMA size 2 was inserted after induction with dilute propofol via a Hickman catheter.² Anaesthesia was maintained with nitrous oxide and isoflurane in oxygen. The airway was well maintained and the child woke rapidly and pleasantly, with no sign of distress.

We elected to use this technique for daily radiotherapy to avoid daily intubation with its possible morbidity. Pulse

oximetry and closed circuit television were used as monitoring. Five further treatments were completed uneventfully using this technique and the child's confidence became such that he no longer required anaesthesia for the remaining sessions of radiotherapy.

This method proved to be an acceptable alternative to our standard technique for CT radiotherapy planning and daily radiotherapy. It may become the elective management for CT radiotherapy planning which takes longer than conventional radiotherapy. We would use it again for daily radiotherapy if ketamine gave a further problem.

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Laryngeal mask airway for emergency tracheostomy in a neonate

A girl, 2.75 kg, born at term with severe Pierre-Robin syndrome, was successfully resuscitated with a laryngeal mask, after ventilation of the lungs by facemask and tracheal intubation proved impossible. The laryngeal mask (Brain Airway size 1) was removed shortly when spontaneous breathing commenced.¹

The baby developed airway problems which were managed with a nasopharyngeal airway and a tongue tie over the next few days. The baby's airway obstructed early on the third morning. Intubation again proved impossible and a laryngeal mask was inserted and the airway was resecured.

It was decided that a tracheostomy should be performed because of the severe facial abnormalities, recurrent airway difficulties and the need to secure the baby's airway during an 80-mile transfer to a paediatric centre, for repair of the hare lip.

The baby was taken to theatre with the laryngeal mask in place. The vocal cords were seen through a paediatric fiberoptic bronchoscope, passed down the centre of the laryngeal mask, and confirmed the larynx was normal before anaesthesia was administered.

Anaesthesia was induced with halothane in 100% oxygen via an Ayre's T piece connected to the laryngeal mask. An unsuccessful attempt was made to insert a bougie blindly down the laryngeal mask into the larynx when the depth of anaesthesia was sufficient. Laryngoscopy was then attempted (the laryngeal mask was removed); when neither the vocal cords nor the epiglottis were seen tracheal intubation was not attempted, and the laryngeal mask was reinserted. A tracheostomy was successfully performed whilst the anaesthetic was administered through the laryngeal mask. Two days later the baby was transferred to

the regional paediatric centre where her hare lip was successfully repaired.

The combination of hare lip, cleft palate and micrognathia seen in the Pierre-Robin syndrome present notoriously difficult airway problems.² The airway can usually be managed after birth by a combination of posture, insertion of tongue ties and nasopharyngeal airways. The breathing difficulties begin to resolve within a month as the baby grows. Usually the anaesthetist does not encounter these patients until they present for surgical repair of the cleft palate at about 6 months of age; intubation can be impossible even at this age and occasionally requires tracheostomy.

Successful anaesthetic management without the use of the laryngeal mask in this patient would have been extremely difficult. The airway enabled an unhurried tracheostomy to be performed.

The role of the laryngeal mask in neonatal anaesthesia clearly needs to be evaluated. There are three questions: should it replace awake intubation; what operations are most suited to its use; does it have a place in resuscitation and interhospital transfer?

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Fiberoptic intubation and double-lumen tubes

Intubation of the trachea aided by the fiberoptic laryngoscope may be facilitated by using smaller diameter endotracheal tubes than might otherwise be employed, and by choosing the nasal route. When a double-lumen tube is indicated for the purposes of anaesthesia for pulmonary surgery, these additional aids become unfeasible.

I was recently faced with the prospect of anaesthetising a patient for thoracotomy, who was known to be difficult to intubate.

A left-sided disposable Mallinckrodt double-lumen tube was threaded over the fiberoptic laryngoscope, and anaesthesia was induced with propofol followed by suxamethonium. A Macintosh laryngoscope was passed into the pharynx; with optimal positioning, the tip of the epiglottis was just visible. The end of the fiberoptic instrument was then positioned behind the epiglottis, under direct vision. The Macintosh laryngoscope was then held by an assistant, and the glottis was viewed through the fiberoptic instrument. The latter was advanced into the trachea with a minimum of manipulation, and it was then a simple matter to insert the double-lumen tube.

The combined use of a Macintosh laryngoscope with a fiberoptic laryngoscope in this way has the advantage that it permits the anaesthetist to present the instrument near to the glottis under direct vision with ease and rapidity, and incidentally without the necessity for the considerable degree of skill in manipulation of the fiberoptic instrument, which its optical characteristics and innate orientational uncertainties impose.

This combined method for double-lumen intubation seems likely to be helpful where difficulty is anticipated; it might be proposed further that where anaesthetists have not yet gained expertise with the fiberoptic instrument, or where nasal intubation is contraindicated, the combined technique may make oral intubation in difficult cases easier than either technique on its own.

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Pulmonary artery catheter occlusion

We wish to report a problem which we have encountered on two occasions when using a balloon-tipped pulmonary artery catheter.

Shortly after the successful introduction of a balloon-tipped thermodilution catheter (Spectramed Pentacath 7.5 FG) it was noted that the pulmonary artery pressure

trace, measured through the distal lumen of the catheter, had become damped. Considerable resistance was encountered when attempts were made to flush this lumen and the other two proximal lumens of the catheter. It was apparent on close examination that the locking nut on the catheter positioning sleeve had been overtightened and was compressing the catheter, with the result that the lumina had become partially occluded. The quality of the pressure trace improved and the resistance to injection decreased when the nut was loosened.

It was not clear whether this is a problem with the particular introducer and positioning sheath we used (Peter

Von Berg Medizintechnik Cat. No. EKV-8-K-JS) or a more general problem, but it is obvious that care needs to be exercised since the signs are similar to those which would be caused by partial occlusion of the lumina of the catheter by blood clot and might lead to premature and unnecessary removal of the catheter. We suspect that problems with previous similar catheters in this unit may have been due to the same cause.

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Drug usage by anaesthetists

We read with interest the letter from Dr Mirakhur (*Anaesthesia* 1990; **45**: 500-1). Any information concerning the activities of our colleagues is always of interest. It is a pity, therefore, that his survey should have attracted such a poor response rate (44.8%). This must impose difficulties in determination of the validity of any data derived from this study since nonresponders (the majority in this survey) are known not to be typical of the population,¹ and their behaviour cannot necessarily be assumed from presented data.

Since none of the authors viewed the initial survey, we cannot determine if the drug usages cited are the drugs most commonly used by the responders in their own recollection or if some other means were used to prevent reporting biases derived from imprecise recall.

We also have recently collected data on drug usage in our hospital in the course of a prospective study on awareness including 1000 anaesthetics. None of the anaesthetists involved was aware of the study, and the data should consequently represent their actual practice. The survey specifically excluded obstetric and paediatric patients, those undergoing intracranial surgery or who were unable to communicate for whatever reason, and those patients discharged before the interview time (20-36 hours after operation). This latter criterion resulted in the elimination of all day-case anaesthetics from our survey. The primary conclusions of this study are to be published separately, but the results of the examination of anaesthetic techniques are shown in Table 1.

The exclusion of certain patient groups alters the patterns of drug usage observed, and we would expect, for example, the frequency of use of halothane to be lower than Dr Mirakhur's data might predict (249 administrations in our study; Dr Mirakhur's data would lead us to have expected 358). The frequency of use of other volatile agents is higher than expected for similar

reasons; enflurane was administered 517 times, isoflurane 209 times and other agents used 14 times. This increased predominance of enflurane may reflect the fact that drug price lists have been a prominent feature in our anaesthetic rooms for several years, although this does not explain the high usage (169 occasions) of propofol.

Dr Mirakhur suggested that 89% of his respondents would use suxamethonium in adults if the occasion demanded it. It was used in our study on 111 occasions to facilitate tracheal intubation. This would confirm a willingness to use a useful drug despite known problems, and confirms its place in current clinical practice.

Our data confirm the dominance of the newer short-acting neuromuscular blocking agents, with a much lower usage of alcuronium than that reported by Dr Mirakhur, and only one use of pancuronium in our 1000 anaesthetics.

The widespread dominance of fentanyl as an analgesic surprised us. The use of fentanyl was only 55% of that reported by Dr Mirakhur. Similarly there was a dominance of papaveretum as an analgesic in our study; it was given on 435 occasions. This was only partly as a consequence of the popularity of papaveretum and hyoscine premedication. Alfentanil was commonly used at induction, presumably for its ability to obtund the cardiovascular response to laryngoscopy.

Cardiostability, in addition to a possibly better immune response profile, also accounted for the higher incidence of use of etomidate (95 occasions).

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Reference

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Table 1. Number of administrations of various anaesthetic drugs in 1000 anaesthetics.

Induction agents		Muscle relaxants		Analgesics	
Thiopentone	679	Vecuronium	343	Fentanyl	324
Propofol	169	Atracurium	141	Papaveretum	435
Methohexitone	50	Alcuronium	61	Morphine	9
Etomidate	95	Pancuronium	1	Alfentanil	149
Others	6	Suxamethonium only	111	Pethidine	25
		Others	33	Local anaesthesia	81

Obstruction in a pressure line

A dangerous malfunction of the Engström Erica ventilator occurred after cleaning and autoclaving of the patient system.

A patient was being weaned from ventilatory support using the Erica when he suddenly experienced severe respiratory difficulties. He was found to be cyanosed with an overinflated chest, with ventilation arrested in inspiration. The ventilator registered an airway pressure of 12.0 kPa and the alarm was triggered. The patient was immediately disconnected from the machine and the lungs were ventilated manually which caused an immediate improvement. Examination and chest radiograph excluded a pneumothorax. A fault of the ventilator was presumed and an alternative was substituted.

The patient system was removed, stripped and inspected and the cause readily identified. A foreign body was discovered in the fine-bore tubing which conducts gas from the machine to a rubber diaphragm which occludes the expiratory port during inspiration. A 'ball-valve' effect of the foreign body maintained the pressure on the diaphragm, and thereby prevented cycling to expiration during a series of breaths. The patient was thus exposed to, and held at, the maximum pressure which had inadvertently been set on the machine at 12 kPa.

The blockage was identified as dried 'hibiscrub' which was used for cleaning before sterilisation in the autoclave where it presumably solidified. The manufacturers' instructions recommend that when cleaning is necessary before sterilisation the final rinse should be with water alone and observation of the guidelines might have prevented this occurrence.

This hazard was further compounded by an error in setting the high pressure limit. The pressure cannot be set above 7.0 kPa without depression of a safety override button to attain the higher values. This safety feature had not prevented a wrong setting.

Staff in the Intensive Care Unit, and Medical Physics department have been alerted to this hazard which it is hoped should not occur again.

This report demonstrates how two separate avoidable factors can result in serious risk to a patient. It was fortunate that the rapid response of ICU staff prevented injury, and the patient was subsequently discharged to the ward and home.

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A reply

We are grateful for the opportunity to comment on this incident which serves to re-emphasise the need for all equipment to be used in accordance with manufacturer's recommendations.

This is important in usage, cleaning and, in particular, the correct settings of alarms.

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Failure of nitrous oxide supply

The anaesthetic gas supply at King Edward Memorial Hospital, Falkland Islands, is from a bank with two cylinders each of oxygen and nitrous oxide. A fault developed in the supply of nitrous oxide to the operating theatre, and the alarm system registered a pressure fault. A nitrous oxide cylinder with an internal tube (liquid take-off cylinder, cylinder with dip-stick) was inadvertently connected to the bank. Liquid nitrous oxide escaped through the internal tube in the liquid take-off cylinder, into the inlet manifold and submerged all internal parts of

manifold. This resulted in the failure of regulators causing a pressure fault to the nitrous oxide supply. The cylinder in question was colour-coded as for a gas take off nitrous oxide cylinder.

The manufacturer and origin of this cylinder is not known and the writer would be grateful to receive information about them.

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Anaesthesia, 1990, Volume 45, page 897

Erratum

Anaesthesia, 1990, Volume 45, page 591-2

Confirmation of tracheal intubation in a neonate using the Fenem CO₂ detector

The Editor omitted the name of Dr A. Lloyd-Thomas, FFARCS, as one of the authors of this communication to whom we offer our apologies.

Anaesthetic literature

This section of *Anaesthetic literature* contains references from *Current Contents—Life Sciences* for June 1990. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase III, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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- Pathophysiology of cancer pain. PAYNE R. In: FOLEY KM *et al.* eds. *Proceedings of the 2nd International Congress on Cancer Pain*. NY: Raven Press 1990: 13.

Treatment and medication

- Nonsteroidal anti-inflammatory analgesics in cancer pain. BEAVER WT. In: FOLEY K *et al.* eds. *Proceedings of the 2nd International Congress on Cancer Pain*. NY: Raven Press 1990: 109.
- Anesthetic approaches in cancer pain. COUSINS MJ. In: FOLEY K *et al.* eds. *Proceedings of the 2nd International Congress on Cancer Pain*. NY: Raven Press 1990: 249.
- A controlled trial of transcutaneous electrical nerve stimulation (TENS) and exercise for chronic low back pain. DEYO RA, WALSH NE *et al. New England Journal of Medicine* 1990; 322: 1627.

- Opioid analgesic therapy in cancer pain. INTURRISI CE. In: FOLEY K *et al.* eds. *Proceedings of the 2nd Internat. Congress on Cancer Pain*. NY: Raven Press 1990: 133.
- Pharmacokinetic-pharmacodynamic relationships of methadone infusions in patients with cancer pain. INTURRISI CE, PORTENOY RK *et al.* *Clinical Pharmacology and Therapeutics* 1990; **47**: 565.
- Morphine and oxycodone hydrochloride in the management of cancer pain. KALSO E, VAINIO A. *Clinical Pharmacology and Therapeutics* 1990; **47**: 639.
- Benzodiazepines and chronic pain. KING SA, STRAIN JJ. *Pain* 1990; **41**: 3.
- Management of postoperative pain: review of current techniques and methods. LUTZ LJ, LAMER TJ. *Mayo Clinic Proceedings* 1990; **65**: 584.
- Continuous intravenous infusion of opioid drugs in the management of cancer pain. PORTENOY RK. In: FOLEY K *et al.* eds. *Proceedings of the 2nd International Congress on Cancer Pain*. NY: Raven Press 1990: 219.
- Spinal analgesia—a modern approach to the treatment of severe pain. ROSENBERG PH. *Journal of Internal Medicine* 1990; **227**: 291.
- Adjuvant analgesic therapy in cancer pain. WALSH TD. In: FOLEY K *et al.* eds. *Proceedings of the 2nd International Congress on Cancer Pain*. NY: Raven Press 1990: 155.

Other

Treatment and medication

- Mechanisms by which mitochondria transport calcium. GUNTER TE, PFEIFFER DR. *American Journal of Physiology* 1990; **258** (No. 5 Part 1): C755.

Obituaries

- Cronin, M.M.**, MB, BCh, BAO, FFARCS, formerly Consultant Anaesthetist, St Bartholomew's Hospital. Qualified Dublin in 1958.
Hain, W.R., MB, BS, MRCS, LRCP, FFARCS, formerly Consultant Paediatric Anaesthetist, Nottingham. Qualified University of London in 1962.
Houldsworth, Sir (H.) Basil Bt., MRCS, LRCP, DA, FFARCS, formerly Consultant Anaesthetist, Barnsley. Qualified University of Leeds in 1946.
Knowles, G.S.A., MB, ChB, DA, FFARCS, formerly Consultant Anaesthetist, Wakefield. Qualified University of Leeds in 1929.
Rollason, W.N., MB, BS, MRCS, LRCP, DA, FFARCS, formerly Consultant Anaesthetist, Aberdeen. Qualified University of Birmingham in 1940.
Sinclair, R.N., MB, ChB, MD, DA, FFARCS, formerly Consultant Anaesthetist, Glasgow. Qualified University of Glasgow in 1938.
Wood Smith, F.G., MA, MB, BChir, MRCS, LRCP, DA, FFARCS, formerly Consultant Anaesthetist, Hammersmith Hospital. Qualified University of Cambridge in 1931.

International congress calendar

1990

- 1-3 October.** London. *International Meeting jointly sponsored by The Royal Society of Medicine and The New York Academy of Sciences. Advances in the understanding and treatment of asthma.*
Information: Fiona Morris, The Royal Society of Medicine, 1 Wimpole Street, London, W1M 8AE.
- 8-10 October.** Rotterdam. *Eleventh International Symposium on Information Technology in Anesthesia, Intensive Care and Cardiopulmonary Medicine.*
Information: Dr Omar Prakash, Chief, Thorax Anesthesia, Thorax Centre, Erasmus University, 3000 DR Rotterdam, The Netherlands.
- 13 October.** London. *Postgraduate Study day with the College of Anaesthetists.*
Information: College of Anaesthetists, 35 Lincoln's Inn Fields, London, WC2.
- 19-23 October.** Las Vegas. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 23-31 October.** Bristol. *Clinical Anaesthesia in the 1990s.*
Information: British Council, 65 Davies Street, London W1Y 2AA, UK.
- 24-27 October.** Salzburg. *1st annual meeting of European Society for Computing and Technology in Anaesthesia and Intensive Care.*
Information: Dr L. Moser, Anaesthesiologie, Landers-krankenhaus, Müllner Hauptstrasse 48, A5020 Salzburg, Austria.
- 31 October-2 November.** Mainz. *International Symposium. Echocardiography.*
Information: Professor Dr Raimund Erbel, 11. Medical Clinic, Johannes Gutenberg-University Mainz, Langenbeckstrasse 1, D-6500 Mainz, West Germany.
- 3-4 November.** New York. *28th Annual Bernard H. Eliasberg Memorial Symposium.*
Information: Anita Guffin, MMS, Mount Sinai Medical Center, One Gustave Levy Place, New York, NY 10029, USA.
- 5-10 November.** Sao Paulo. *36th Brazilian Congress of Anesthesiology.*
Information: Dr R. Mathias, Rua Caiubi 666, Sao Paulo, Brazil 05010.
- 7-9 November.** Israel. *Fourth International Symposium of Anaesthesia and Intensive Care.*
Information: Dr G. Gurman, Division of Anaesthesiology, Soroka Medical Centre, Beer-Sheva 84101, Israel.

- 8-9 November.** Pakistan. *Third International Anaesthesia Conference.*
Information: Dr Jamila Bilal, Associate Professor, Secretary Organising Committee, Khushal Khan Road, F/21 Peshawar U/Town, Pakistan.
- 18-21 November.** Kyoto, Japan. *4th International Symposium on the Pain Clinic.*
Information: 4th WSPC Secretariat, Department of Anesthesiology, Osaka Medical College, c/o Inter Group Corp., Shohaku Bldg., 6-23 Chayamachi Kita-ku, Osaka 530, Japan.
- 21-24 November.** Trieste. *5th postgraduate course on Recent Advances in Anaesthesia, Pain, Intensive Care and Emergency.*
Information: Trieste Traduzioni Congressi, Viale XX Settembre 4, 34125 Trieste, Italy.
- 26-29 November.** Rotterdam. *Sixth international symposium on Cardiopulmonary Urgencies and Emergencies.*
Information: Dr O. Prakash, Thorax Anesthesia, Erasmus University, 3000 DR Rotterdam, The Netherlands.
- 5-9 December.** San Juan. *15th Caribbean Symposium in Anaesthesia and Related Fields.*
Information: Miguel Colon-Morales, GPO Box 4547, San Juan, Puerto Rico 00936.
- 8-12 December.** New York. *Forty-fourth Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.
- 15-22 December.** Vail. *Current Issues in Medicine.*
Information: Professional Seminars, P.O. Box 012318, Miami, FL 33101, USA.
- 27-30 December.** Madras. *39th Annual Conference Indian Society of Anaesthetists.*
Information: Dr K. Balakrishnan, Organising Secretary, Department of Anaesthesia (Specialty), Government General Hospital, Madras 600 003, India.

1991

- 10-13 January.** Miami. *28th Annual Post-Graduate Seminar in Anesthesiology.*
Information: Barbara McNulty, Continuing Education Programs, 7480 Fairway Drive, Suite 106, Miami Lakes, Florida 33014, USA.

- 12–19 January.** Barbados. *9th Annual Symposium: clinical update in Anesthesiology.*
Information: Ms H. Phillips, Mount Sinai Medical Center, 1 Gustave L. Levy Place, Box 1010, New York, NY 10029, USA.
- 15–17 January.** Doha-Qatar. *First Gulf Conference on Intensive Care Medicine.*
Information: Dr Jamal S. Al-Shanableh, Consultant Cardiac Anesthesiologist, Secretary, P.O. Box 3050, Hamad General Hospital, Doha-Qatar.
- 17–19 January.** London. *Winter Scientific Meeting and Technical Exhibition Queen Elizabeth Conference Centre.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2–9 February.** Colorado. *17th Annual Vail Conference in Anesthesiology.*
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.
- 9–16 February.** Vail. *16th Annual Symposium in Intensive Care.*
Information: Professional Seminars, P.O. Box 012318, Miami, FL 33101, USA.
- 8–12 March.** San Antonio. *65th Congress of the International Anesthesia Research Society.*
Information: Emerson A. Moffitt, IARS, 3645 Warrensville Center Road, Cleveland, Ohio 44122, USA.
- 9–14 March.** Pretoria. *The 1991 National Anaesthetic Congress of the South African Society of Anaesthetists.*
Information: Professor J.M. Hugo, Chairman, Department of Anaesthetics, Faculty of Medicine, P.O. Box 667, Pretoria, South Africa.
- 28–30 March.** Osaka. *38th Annual Meeting of the Japan Society of Anesthesiology.*
Information: Professor M. Fujita, Japan Society of Anesthesiology, TY Building, 18–11 Hongo 3-chome, Bunkyo-Ku, Tokyo 113, Japan.
- 4–7 April.** Cincinnati. *16th Annual Meeting of the American Society of Regional Anesthesia.*
Information: P.O. Box 11086, Richmond, Virginia 23230–1086, USA.
- 3–5 April.** Oxford. *Junior Anaesthetists' Group of the Association of Anaesthetists of Great Britain and Ireland Linkman Conference and Annual Scientific Meeting.*
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 15–18 April.** Hamamatsu, Japan. *6th International Symposium on Computing in Anesthesia and Intensive Care.*
Information: Dr K. Ikeda, Chairman of the Organising Committee, c/o Department of Anesthesiology, Hamamatsu University School of Medicine, 3600 Handa-cho, Hamamatsu, Shizuoka, 431–31 Japan.
- 17–21 April.** Antilles. *19th International Society on Oxygen Transport to Tissue.*
Information: Professor W. Erdmann, Department of Anesthesiology, Dr Molewaterplein 40, 3015 GD Rotterdam, The Netherlands.
- 18–21 April.** Paris. *European Academy of Anaesthesiology. Refresher Course.*
Information: Professor J.M. Desmonts, Department d'Anesthésie, Hôpital Bichat, 46 rue Henri-Huchard, 75018 Paris, France.
- 23–27 April.** Montreal. *Second International Symposium on Pediatric Pain.*
Information: Pain Secretariat, 3450 University Street, Montreal, Quebec, H3A 2A7, Canada.
- 3–5 May.** Philadelphia. *AUA Annual Meeting.*
Information: Stephen J. Prevotnik, Department of Anesthesia, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104, USA.
- 4–8 May.** San Antonio. *Society of Cardiovascular Anesthetists.*
Information: P.O. Box 11086, Richmond, Virginia 23230–1086, USA.
- 9–12 May.** Washington DC. *6th International Dental Congress on Modern Pain Control.*
Information: American Dental Society of Anesthesiology, Inc., 211 E. Chicago Avenue, Suite 948, Chicago, IL 60611, USA.
- 24–25 May.** The Netherlands. *Receptors of the Brain, Lung and Heart: State of the Art.*
Information: Cader Research B.V., P.O. Box 85, 4854 ZH Breda/Bavel, The Netherlands.
- 27–31 May.** Montreal. *McGill University Annual Review Course in Anaesthesia.*
Information: Post Graduate Board, Royal Victoria Hospital, 687 Pine Avenue West, Room H308, Montreal Quebec, H3A 1A1, Canada.
- 21–25 June.** Quebec City. *48th Annual Meeting of Canadian Anaesthetists' Society.*
Information: Ms Ann Andrews, CAS, 187 Gerrard St. E., Toronto, Ontario M5A 2E5, Canada.
- 11–13 September.** Harrogate. *Linkman and Annual Scientific Meeting of Association of Anaesthetists of Great Britain and Ireland.*
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 11–15 October.** Baghdad. *4th Pan-Arab Congress of Anaesthesia and Intensive Care.*
Information: Dr M. Keilani, P.O. Box 17078, Amman, Jordan.
- 26–30 October.** San Francisco. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 7–11 December.** New York. *Forty-fifth Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

1992

- 1–8 February.** Colorado. *18th Annual Vail Conference in Anesthesiology.*
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.
- 13–17 March.** San Francisco. *66th Congress of the International Anesthesia Research Society.*
Information: International Anesthesia Research Society, 3645 Warrenville Center Road, Cleveland, Ohio 44122, USA.
- 25–29 March.** Tampa. *17th Annual Meeting of the American Society of Regional Anesthesia.*
Information: P.O. Box 11086, Richmond, Virginia, 23230–1086, USA.
- 29 March–2 April.** Atlanta, Georgia. *The Third International Symposium on the History of Anaesthesia.*
Information: R.K. Calverley, Medical Center, University of California, 225 Dickinson Street, San Diego, California CA 92103, USA.
- 1–3 April.** Bristol. *Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2–6 May.** Boston. *Society of Cardiovascular Anesthetists.*
Information: P.O. Box 11086, Richmond, Virginia, 23230–1086, USA.
- 4–9 June.** Toronto. *49th Annual Meeting of the Canadian Anaesthetists' Society.*
Information: 187 Gerrard Street E, Toronto, Canada M5A 2E5.
- 7–12 June.** Barcelona. *Anesthesia 92.*
Information: Pacifico, S.A.: c/Muntaner, 112 08036-Barcelona, Spain.
- 10–13 June.** Brussels. *European Society of Regional Anaesthesia (UK) Meeting.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 12–19 June.** The Hague. *10th World Congress of Anaesthesiology.*
Information: Dr Harm Lip, Nilantweg, 99, 8041 AR Zwolle, Netherlands.
- 9–11 September.** Bournemouth. *Linkman and Annual Scientific Meeting.*
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 17–21 October.** New Orleans. *American Society of Anesthesiologists Annual Meeting.*
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.

12–16 December. New York. *46th Postgraduate Assembly in Anesthesiology.*
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists, Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

1993

12–16 February. Utah. *38th Annual Postgraduate Course in Anesthesiology — 'Anesthesiology: Today and Tomorrow'.*
Information: Vicky Larson, Department of Anesthesiology, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, Utah 84132, USA.

29 April–2 March. North Carolina. *Meeting of the Association of University Anesthetists.*

Information: Francis M. James III, Department of Anesthesia, Wake Forest University Medical Center, 300 S. Hawthorne Road, Winston-Salem, North Carolina 27103, USA.

1–4 September. Liverpool. *European Course and Congress in Paediatric Anaesthesia.*

Information: Dr P.D. Booker, Alder Hey Hospital, Liverpool L12 2AP.

22–24 September. Glasgow. *Linkman Conference and Annual Scientific Meeting.* Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland and the Canadian Anaesthetists' Society.

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

9–13 October. Washington DC. *American Society of Anesthesiologists Annual Meeting.*

Information: Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068, USA.

1994

7–9 September. Brighton. *Linkman Conference and Annual Scientific Meeting.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

2–7 October. Jerusalem. *European Congress of Anaesthesiology.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

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Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK.

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The names of the authors should be typed IN CAPITALS across the title page immediately beneath the titles without degrees or designations. Initials should precede the surname. If there is more than one author the word 'AND' should be placed before the name of the last author.

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The second page should carry a summary of not more than 150 words. The summary should state the purpose of the study or investigation, basic procedures, main findings and their statistical significance, and the principal conclusions.

Do not use abbreviations except for units of measurement (e.g. mg, cm, etc.).

Key (indexing) words. Below the abstract, provide and identify as such, three to 10 key words or short phrases that will assist indexers. Use terms from the Medical Subject Headings list from *Index Medicus*. The Editor may modify these at proof stage to conform with agreed practice of certain other anaesthetic journals in the English language.

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Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables and legends by arabic numerals. References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration. Use double or treble spaced typing.

Use the form of reference adopted by the US National Library of Medicine and used in *Index Medicus*. Use the style of the examples cited at the end of this section.

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The references must be verified by the author(s) against the original documents.

(continued overleaf)

Examples of correct form of references

Note: first and last pages in all references.

JOURNAL

Standard journal article—(List all authors)

SOTER NA, WASSERMAN SI, AUSTEN KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *New England Journal of Medicine* 1976; **294**: 687–90.

Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

WEINSTEIN L, SWARTZ MN. Pathogenic properties of invading micro-organisms. In: SODEMAN WA Jr, SODEMAN WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*. No. 69) [DHEW publication No. (HSM) 72-1036].

OTHER ARTICLES

Newspaper article

SHAFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

Magazine article

ROUECHE B. Annals of medicine: the Santa Claus culture. *The New Yorker* 1971 Sept 4: 66–81.

TABLES

Do not include tables in the text. Start a new sheet for each table and space the material adequately. The author(s) name(s) should appear in the top right-hand corner.

Indicate the approximate position of each table in relation to the subject matter of the text in the left-hand margin of the appropriate page on the manuscript. Do not submit tables as photographs. Number tables consecutively with arabic numerals. Supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes. Explain in footnotes all non-standard abbreviations that are used in each table. For footnotes, use the following symbols in this sequence: *, †, ‡, §, ||, ¶, **, ††, etc. Identify statistical measures of variations such as SD and SEM. Legends for tables should appear on the face of the table.

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Type legends for illustrations double spaced with arabic numerals corresponding to the illustrations. When symbols, arrows, numbers or letters are used to identify parts of the illustrations, identify and explain each one clearly in the legend.

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Statistics and measurements should be given in figures except that numerals one to nine should be in words if not followed by a measurement symbol (e.g. 'two patients' but 2.0 mg). The *Système International* (SI) will usually be used except that vascular pressures will be recorded in mmHg and cmH₂O. Imperial measurements will not be used except in an historic context. The 24 hour clock will be used.

Contributors are advised to study the SI *Conversion Tables* provided in the January 1978 issue of *Anaesthesia, Units, Symbols and Abbreviations* (A Guide for Biological and Medical Editors and Authors) published by the Royal Society of Medicine, London W1M 8AE, and *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1**: 532–5).

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Should be addressed to Dr J. N. Lunn, Editor of *Anaesthesia*, Department of Anaesthetics, University Hospital of Wales, Heath Park, Cardiff CF4 4XW, UK.

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REVIEW JOURNALS

This journal is covered by *Current Contents*, *ASCA*, the *Science Citation Index* and *Index Medicus*.

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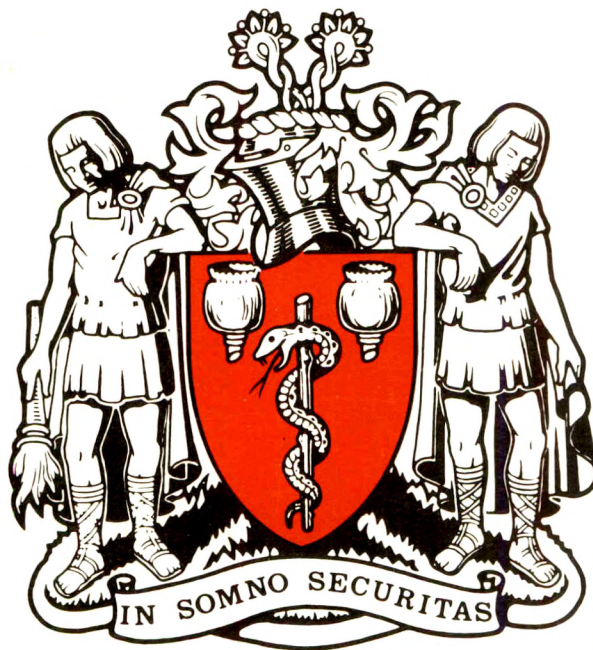
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Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 45 Number 11 November 1990



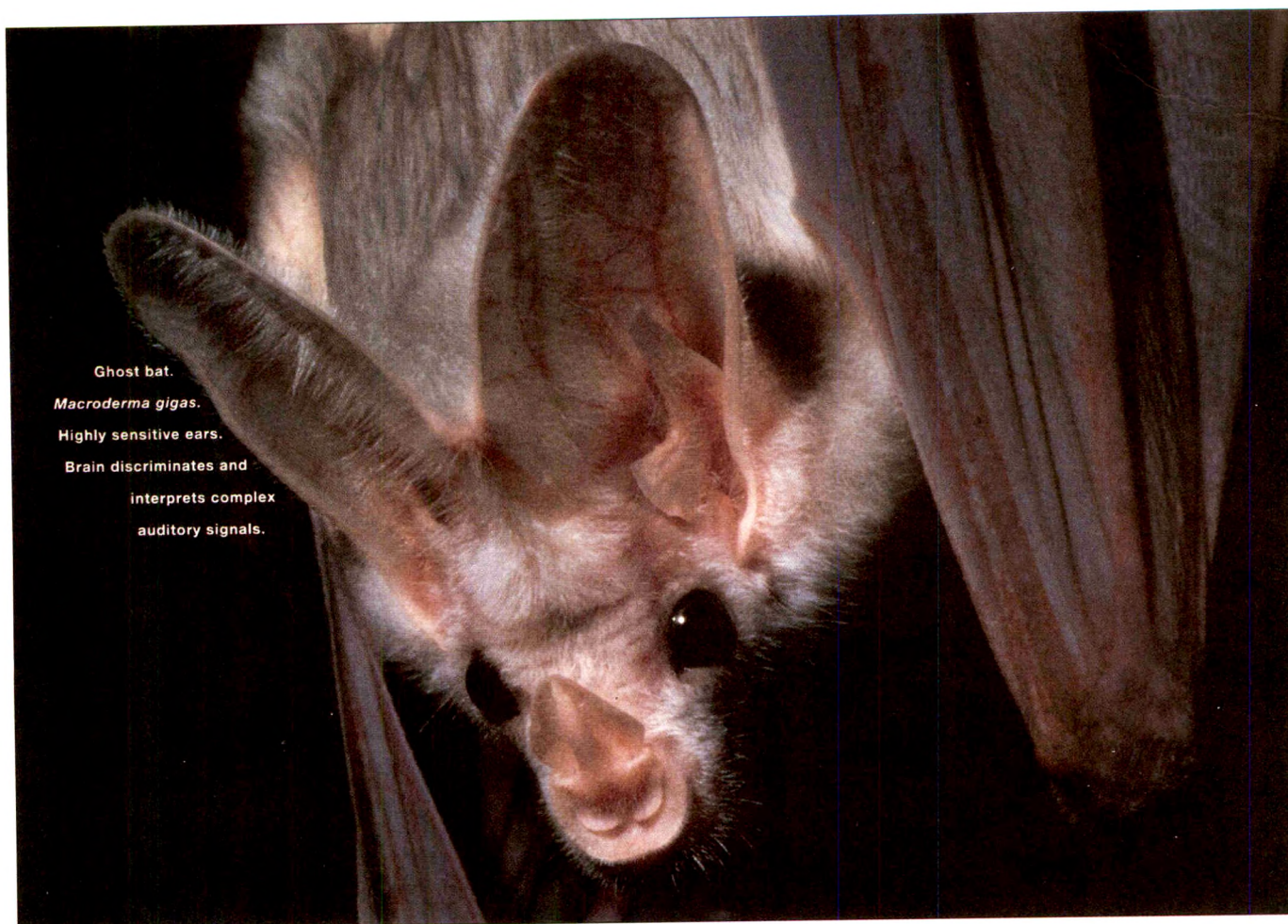
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Anaesthesia

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ANAESTHESIA: ISSN 0003-2409, Volume 45 1990, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. **All advertising** enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293).

Annual subscription price including postage: £118 UK and US \$228 overseas. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel. 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

Anaesthesia is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

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▼ Prescribing Information

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Dosage and Administration: Dopacard must be diluted before use and administered intravenously via a cannula or catheter into a central or large peripheral vein. Full instructions are given in the data sheet and package insert. **Preparation** The contents of four ampoules should be injected aseptically into one of the following: 0.9% Sodium Chloride Injection or 5% Dextrose Injection (500 or 250ml). **Recommended dosage for adults including the elderly:** Infusion should begin at a dose of 0.5 µg/kg/min and may be increased to 1 µg/kg/min and then in increments (1 µg/kg/min) up to 6 µg/kg/min at 10-15 minute intervals. The rate of administration and duration of therapy should be adjusted according to the patient's response as determined by heart rate, blood pressure, urine output and, if possible, measurement of cardiac output.

Contraindications, Precautions,

Warnings: Contraindications Concurrent MAOI administration, left ventricular outlet obstruction (such as hypertrophic obstructive cardiomyopathy or aortic stenosis), phaeochromocytoma, thrombocytopenia. **Precautions** If any correction of hypovolaemia is required this should be achieved before the administration of Dopacard. **Warnings** Dopacard should be administered with caution to patients with acute myocardial infarction or recent episodes of angina pectoris. A fall in circulating platelet numbers has been observed in some patients. No adverse experiences attributable to alterations in platelet count have been seen in clinical studies. Plasma potassium may decrease and blood glucose may increase during Dopacard administration and care is required in its use in patients with, or at risk of, hypokalaemia or hyperglycaemia. There is no evidence to suggest that Dopacard has significant arrhythmogenic potential. However, if a cardiac arrhythmia occurs during administration a reduction or temporary discontinuation of the infusion should be considered. The safety and efficacy of Dopacard for use in children has not been established.

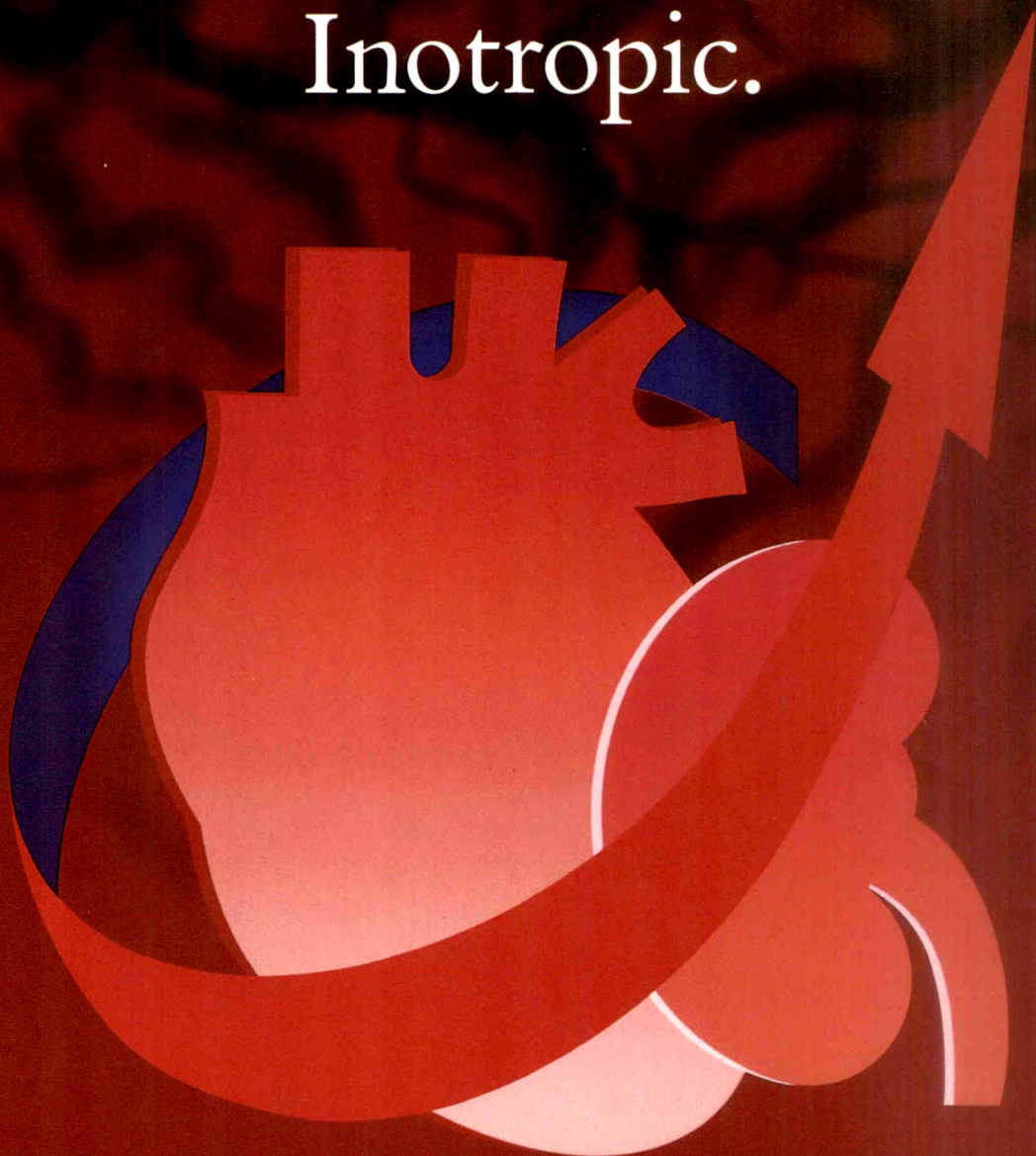
Use in Pregnant and Lactating Women Dopacard is not currently recommended for use in pregnant and lactating women. **Side effects** Increases in heart rate may occur during infusion of Dopacard; in most cases these are not clinically significant. Occasionally excessive tachycardia or ventricular ectopic beats have been noted during the infusion, necessitating reduction or temporary discontinuation of the infusion. Tachycardia may be more pronounced in patients with pre-existing atrial fibrillation. The following side-effects have been reported infrequently, in most cases at high dosage: nausea, vomiting, anginal pain and tremor. **Interactions** Dopacard may potentiate the effects of exogenous noradrenaline or dopamine. Concomitant use of β₂-adrenergic and dopamine receptor antagonists may cause attenuation of the pharmacological effects of Dopacard.

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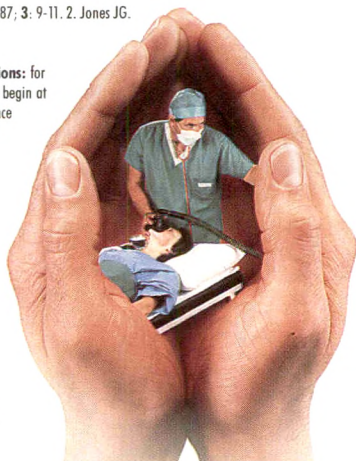


References 1. Hargrove RL. J Med Def Union 1987; 3: 9-11. 2. Jones JG. Anaesthesia Rounds, ICI plc, 1988; No. 21.

Prescribing Information: Isoflurane. **Indications:** for inhalation anaesthesia. **Dose:** Induction should begin at 0.5% and be adjusted appropriately. Maintenance concentrations generally lie between 1.0% and 2.5%. For caesarian section, 0.5-0.75% Isoflurane in a mixture of oxygen/nitrous oxide is recommended. Elderly: lesser concentrations normally required.

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Editorial

Ethics and research in anaesthesia

Many doctors find the process and obligation to obtain approval for their research from an ethics committee irritating and annoying. Why should this be so? Part of the problem is that they believe that ethics are merely a matter of personal conscience, that there are no hard and fast rules, and that they are as qualified as the next person to judge what is right or wrong, or scientifically sound. Another part is that research has become more and more the means to improve *curriculum vitae* and augment promotion prospects: obstacles to these ends are resented. Are these feelings justified? First, are there hard and fast principles? Yes, the fundamental concepts are those of autonomy, justice, beneficence, and non-maleficence. These ethical precepts need translation into medical parlance. Autonomy is the first and most important principle, stated strongly in the Nuremberg Code¹ that 'the voluntary consent of the human subject is absolutely essential'. Justice requires us to treat persons fairly, generally in this context in terms of the distribution of burdens and benefits. Beneficence is the obligation to secure the well-being of individuals, and develop our capacity to do better in future. Nonmaleficence is the slightly oversimplified Hippocratic concept of *primum non nocere*. The ramifications of the double-blind placebo-controlled trial can now be approached, with these concepts clear.

Many believe that the Nuremberg code, developed as it was from the revelations about Nazi war crimes, has little to do with 'proper' medical research, but the links are of interest. The code suggests that criminal medical experiments started with the outbreak of the war as a planned part of the Nazi war effort. Many of these atrocities started in fact on the accession of Hitler to power. The wartime 'experiments' were often scientifically crude and inconclusive,² but they characterised a utilitarian and pragmatic approach to research that was not confined to wartime Germany, but was also found elsewhere, and later, Henry Beecher, then Professor of Anaesthesia at Harvard, quoted examples of unethical studies that were undertaken by major institutions with government funds and published in leading journals.³

The Nuremberg Code gave rise to the Declaration of Helsinki, and research protocols often state that it will be followed. Many researchers are unfamiliar, one suspects, with its contents and the following may be of particular relevance to the aspiring researcher. The research must be scientifically sound, and considered by an independent committee who may comment and provide guidance: no autonomy for the researcher! The importance of the objective must be in proportion to the inherent risk to the subject, which has to be carefully assessed beforehand. Every patient, including controls, in any medical study where a disease or illness is being treated, 'should be assured of the best current diagnostic and therapeutic methods'. Consequently, studies should neither use inactive nor placebo treatment, if there is a current active form of therapy. One of the problems can be that the researcher, or more commonly his colleagues, may believe fervently that the new method that

has just come along is the only one that should be used, and that a trial would be unethical. This is not so if the rest of the world is unconvinced. For example, the researcher who believes he has the only answer to postoperative pain is obliged to maintain his scientific 'equipoise', and believe that there is currently no way of telling if the new method is better than the old, until he has generated sufficient robust data to substantiate his belief to the satisfaction of the scientific community at large. There is no doubt that any proposed study should be scientifically sound: to subject patients to worry and discomfort with no prospect of completion, or a satisfactory answer to the hypothesis, is wrong. This also implies that the project must be well supervised, and there must be sufficient time and resources to ensure it is completed. Rotational training schemes are not conducive to this, unless planning is exemplary.

How in practice are research projects considered? In 1975, the Department of Health and Social Security (DHSS) issued advice on the formation of ethics of research committees (HSC(IS)153, June 1975), but their subsequent implementation was haphazard. More recently, the Royal College of Physicians of London has issued guidance,⁴ and research ethics committees were surveyed by the Institute of Medical Ethics,⁵ and debated by Community Health Councils (CHCs).⁶ A draft Department of Health (DoH) circular was recently issued in England and Wales for comment, and a final version may be produced very soon. The draft recommends among other things that the committees should include clinically active doctors and nurses, researchers, and lay people from the CHCs. They should not conduct their business by post, and should consider, among other things, the scientific merit of the project and the payment of the researcher and the subject. The most recent guidelines on research which involves patients⁷ suggests that *per capita* payments to researchers are unethical: payments should be made for a study in its entirety. The draft from the DoH also defines the way the committee should work; it should register and review the progress of the projects it approves. An annual report should be provided for public inspection.

Where does this leave the junior researcher? The study must be scientifically sound, adequately supervised, and respect the wishes of the subject. The result must be worthwhile. If despite this advice, an unethical study is done, and is submitted for publication, what should happen? If all journals turned down such studies, the researchers may stop doing them. However, it may be that some journals by insisting on scientific rigour, may encourage such studies.⁸ What if the data are valuable and important? If the study be rejected, not only might the data be lost, but also the study may be repeated next year by the next ambitious worker. Another approach that is suggested is that the report should be published together with an editorial comment about the study's ethical weakness; the author might well object to this.

Ethical consideration is an important part of the

research process: trainee anaesthetists should have the opportunity to attend a meeting of the local research ethics committee to see how it works (with the understanding that its deliberations are confidential). They should also be encouraged to discuss their research proposals, before they are finalised, with an experienced member of the committee.

Royal Infirmary,
Edinburgh, EH3 9YW

G. B. DRUMMOND

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Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; **1**: 432 5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Recovery after day-case anaesthesia

A 24-hour comparison of recovery after thiopentone or propofol anaesthesia

P. J. HEATH, T. W. OGG AND W. R. GILKS

Summary

Sixty patients who presented for day-case dilatation and curettage were allocated randomly to receive either thiopentone or propofol for induction and maintenance of anaesthesia. One anaesthetist administered all the anaesthetics whilst all assessments were made by one other. The results indicate that early recovery of memory function, critical flicker fusion frequency and subjective feelings of tiredness, drowsiness and alertness were superior in the propofol group. There was a significant difference in subjective feelings of tiredness and drowsiness recorded by the two study groups at 24 hours. Memory function assessed by Wechsler logical memory function passages at 24 hours was impaired in the propofol group in comparison to a group of 'reference' subjects.

Key words

Anaesthesia; outpatient.

Recovery.

It has been established¹ that propofol and thiopentone provide a better quality of induction, maintenance and recovery than the two other commonly used intravenous anaesthetic agents. Immediate recovery after propofol anaesthesia is more rapid than with thiopentone, but there are few reports of longer term differences in recovery between the two agents.^{2,3} Thiopentone is metabolised slowly and is associated with a hangover effect, which is a disadvantage if the agent is used for day-case anaesthesia. The aim of this study was to examine the recovery of patients during the first 24 hours after anaesthesia with propofol or thiopentone.

Method

Approval for this double-blind, parallel-group, comparative study was granted by the local District Ethics Committee. Sixty female patients who presented for dilatation and curettage (D and C) and who satisfied the inclusion criteria took part in the study. All were between 18 and 60 years of age, weighed less than 80 kg and were graded ASA 1 or 2. The patients were accompanied home after the procedure and remained there for 24 hours after recovery. Those taking any other medication were excluded from the study.

Subjects were recruited on arrival at the day surgery unit and gave written informed consent to participate in the study. The National Adult Reading Test was employed as a

measure of Intelligence Quotient at the first testing session only.⁴ All assessments of recovery were performed pre-operatively to establish a baseline, and at 1, 2 and 24 hours after recovery. The 24-hour assessment was made in the patient's home.

A Linear Analogue Rating Scale (LARS) was used to assess tiredness, drowsiness, and alertness.⁵ The 10-cm visual analogue scales were marked 'less tired, less drowsy and less alert' at the left end and 'more tired, more drowsy and more alert' at the right end. Each scale was marked in the centre as an indication of the normal sensation for that time of day. Scores were recorded as millimetres from the left end of the scale.

Patient recovery was assessed using a portable critical flicker fusion apparatus. The mean value of three increasing and three decreasing fusion frequencies was recorded at each test session.

Memory function was assessed by Wechsler logical memory function test passages⁶ and Bethune's modification of the Williams' memory function test. The Wechsler logical memory function test is well established and examines recall for the details of a short story passage immediately after it is read to the subject. There is no visual component to this test. A different Wechsler passage was used at each presentation.

Bethune's modification of Williams' memory function test employs picture cards displaying nine pictures that are

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Accepted 20 February 1990.

Table 1. Mean (SD) values for age, weight, heart rate and intelligence quotient (I.Q.) and duration of anaesthesia in the series.

	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (<i>F</i> -stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
Age; years	43.0 (8.9)	41.3 (8.9)	0.464	42.1 (8.6)	0.758	0.691	0.725
Weight; kg	62.0 (7.4)	61.5 (8.4)	0.798	61.5 (7.5)	0.951	0.795	1.000
Heart rate; beats/minute	72.8 (14.0)	73.1 (13.0)	0.931	70.0 (10.7)	0.535	0.388	0.317
I.Q.	114.6 (6.9)	112.1 (9.2)	0.226	114.6 (5.3)	0.299	1.000	0.202
Duration of anaesthesia; (minutes)	6.3 (1.9)	6.1 (1.7)	0.724	—	—	—	—

given to the subjects for 60 seconds to memorise. The subjects are asked to recall the pictures on the card 10 minutes later. Standard verbal clues are given for those that they are unable to recall and, if still unable to recall any pictures, the subjects are asked to pick the remaining pictures from a card containing the original nine pictures and six distracting pictures. An error score is assigned for the number of pictures that were not recalled at each stage. The maximum error score for each card at each session is 81. Bethune's memory function test was modified further to include a fifth test card, the 'E' card. Subjects were shown two cards at each presentation. The second card was always the 'D' card. One of the remaining cards was presented before the 'D' card at each test session. Penalty scoring for the test was as described originally.⁷

Subjects were allocated randomly to one of the two test groups and were unaware of the anaesthetic agents administered. Both groups received alfentanil 7.5 µg/kg and were allowed to breathe 70% nitrous oxide in oxygen via a Bain system. Anaesthesia was induced in the propofol group with propofol 2.5 mg/kg and maintained with incremental doses of propofol as indicated clinically. One millilitre of lignocaine 1% plain was added to 20 ml propofol to reduce the pain on injection. Anaesthesia was induced in the thiopentone group with thiopentone 5.0 mg/kg and maintained with incremental doses of thiopentone administered as required.

All patients were nursed in a recovery area after surgery and returned to the main ward 30 minutes after recovery from anaesthesia. Intramuscular metoclopramide 10 mg and paracetamol 0.5–1.0 g orally were prescribed as required.

All patients were discharged from the unit within 3 hours of surgery. Two anaesthetists were involved in the study; one administered all the anaesthetics whilst the other, unaware of the agent administered, made all the assessments including those in the patient's home at 24 hours.

A group of 30 volunteers from the nursing and ancillary staff were invited to perform the recovery tests. They served as a reference group and helped to identify any learning effect of the tests employed. We refer to this group as a

reference rather than a control group because in the context of randomised clinical trials the control group is included in the randomisation. The selection criteria for the reference group were similar to the study groups but personnel with prior knowledge of any of the recovery tests were excluded.

Statistical analysis of the data from the two study groups was performed using Student's *t*-test. One-way analysis of variance was employed when the reference group was included in the analysis.¹⁶ Each study group was compared with the reference group using Student's *t*-test. A *p* value of less than 0.05 was considered statistically significant.

Results

Table 1 shows the demographic details and intelligence quotients of patients in the three groups.

Tables 2, 3 and 4 record the LARS scores for tiredness, drowsiness and alertness in the three groups at 0, 1, 2, and 24 hours. A score of 50 is the 'normal' feeling for that time of day for each variable. A score of less than 50 suggests less tired, less drowsy and less alert and scores more than 50 more tired, more drowsy and more alert. Differences between the treatment groups were statistically significant 1 hour after recovery for all these variables and at 24 hours for feelings of tiredness and drowsiness. Drowsiness scores were significantly higher in the thiopentone group than the reference group before the study and alertness scores in both study groups were significantly lower than in the reference group at the pre-operative, and one and 2 hour postoperative test sessions.

Table 5 shows the error scores for Bethune's modification of Williams' memory function test. Higher scores indicate that more errors were made. There was a statistically significant difference between the treatment groups in the performance for new cards one hour after recovery. Both groups performed significantly less well with new cards than did the reference group at one and 2 hours after operation. Patients in the thiopentone group also made more errors than those in the reference group with the 'D' card at one hour.

Table 2. Mean (SD) values for *tiredness* measured by a linear analogue rating scale (mm).

Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (<i>F</i> -stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
0	50.4 (8.5)	50.0 (22.0)	0.920	46.8 (16.4)	0.663	0.290	0.525
1	70.2 (14.4)	57.4 (20.2)	0.007	43.8 (14.7)	<0.0001	<0.001	0.004
2	54.0 (18.0)	52.0 (25.6)	0.732	46.5 (19.1)	0.368	0.123	0.350
24	50.5 (13.4)	39.9 (25.8)	0.050	48.3 (20.3)	0.111	0.622	0.166

Table 3. Mean (SD) values for *drowsiness* measured by a linear analogue rating scale (mm).

Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (F-stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
0	50.9 (9.1)	46.4 (21.9)	0.302	44.6 (14.3)	0.295	0.046	0.708
1	75.4 (11.7)	57.5 (22.6)	<0.001	40.1 (13.7)	<0.0001	<0.001	0.001
2	53.1 (23.5)	50.9 (22.9)	0.719	42.8 (17.4)	0.155	0.059	0.128
24	49.8 (15.1)	36.3 (23.4)	0.010	43.7 (16.8)	0.024	0.145	0.164

Table 4. Mean (SD) values for *alertness* measured by a linear analogue rating scale (mm).

Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (F-stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
0	44.6 (10.5)	40.4 (14.7)	0.204	51.8 (15.8)	0.007	0.042	0.005
1	22.2 (13.5)	33.6 (17.7)	0.007	53.8 (15.6)	<0.0001	<0.001	<0.001
2	41.7 (18.5)	39.6 (18.2)	0.648	53.3 (20.1)	0.013	0.023	0.008
24	49.3 (16.7)	52.3 (25.8)	0.616	53.7 (20.0)	0.734	0.359	0.815

Table 5. Mean (SD) error scores for Bethune's modification of Williams' *memory* function test.

Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (F-stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
New card							
0	12.3 (7.1)	10.9 (7.5)	0.470	11.3 (6.1)	0.732	0.561	0.821
1	30.7 (13.5)	18.9 (10.2)	<0.001	13.0 (8.4)	<0.0001	<0.001	0.018
2	22.1 (12.4)	16.7 (11.0)	0.079	11.7 (6.1)	<0.001	<0.001	0.034
24	15.1 (8.1)	13.6 (8.1)	0.477	11.7 (8.5)	0.283	0.118	0.379
D card							
0	14.0 (7.4)	15.5 (9.4)	0.503	13.5 (9.5)	0.662	0.821	0.416
1	8.7 (6.2)	7.7 (6.2)	0.520	5.3 (4.1)	0.052	0.015	0.082
2	4.8 (5.9)	5.3 (5.3)	0.731	3.2 (4.5)	0.255	0.242	0.103
24	3.9 (4.3)	3.5 (4.3)	0.742	2.4 (4.1)	0.327	0.172	0.315

Table 6. Mean (SD) for the Wechsler *logical memory* passage.

Pieces of information recalled							
Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	Thiopentone v p (F-stats)	Propofol v reference (<i>t</i> -test)	reference (<i>t</i> -test)
0	10.9 (3.9)	11.8 (4.1)	0.390	12.7 (2.8)	0.174	0.045	0.325
1	8.1 (2.4)	8.5 (3.9)	0.607	11.7 (2.5)	<0.0001	<0.001	<0.001
2	12.4 (4.8)	13.3 (5.1)	0.485	14.0 (3.6)	0.397	0.150	0.541
24	12.4 (3.4)	11.2 (4.7)	0.246	14.0 (3.3)	0.018	0.069	0.010

Table 7. Mean (SD) values for the *critical flicker fusion frequency* (Hz) testing in the series.

Hours	Thiopentone	Propofol	p (<i>t</i> -test)	Reference group	p (F-stats)	Thiopentone v reference (<i>t</i> -test)	Propofol v reference (<i>t</i> -test)
0	24.6 (2.7)	23.8 (2.4)	0.215	25.9 (3.2)	0.015	0.094	0.006
1	22.9 (2.5)	23.5 (2.6)	0.351	26.1 (3.6)	0.0001	<0.001	0.002
2	23.6 (2.5)	23.9 (2.0)	0.659	26.1 (3.9)	0.002	0.005	0.008
24	24.9 (3.0)	24.1 (2.8)	0.300	26.1 (3.5)	0.057	0.159	0.018
1-0	-1.7 (2.1)	-0.3 (2.5)	0.024	0.26 (1.6)	0.002	<0.001	0.306
2-0	-1.0 (2.2)	0.1 (2.0)	0.055	0.25 (1.8)	0.038	0.019	0.761
24-0	0.3 (2.3)	0.3 (2.2)	0.987	0.21 (2.0)	0.978	0.872	0.869

Table 6 shows the number of pieces of information recalled from the Wechsler logical memory function passages at each of the test sessions. There were no significant differences between the treatment groups, although significant differences between the reference and treatment groups emerged at 1 and 24 hours. Patients in the thiopentone group recalled less information than those in the reference groups at the pre-operative test session.

Table 7 records the crude critical flicker fusion frequencies for each group at each test session and the mean difference in flicker fusion frequency from the baseline value for each group (Fig. 1). There was a significant difference between the two treatment groups in the difference from baseline at one hour and there was a significant difference between the crude scores for the propofol and reference groups at each test session. Crude scores for the thiopentone group were also significantly different from the reference group at 1 and 2 hours.

Discussion

Day-case surgery is now practised widely throughout the United Kingdom. The importance of rapid, well-maintained recovery with few postoperative sequelae increases as the service expands, and postoperative day patients should not overburden the primary care services. This will be the case only if such patients are discharged when clinically recovered. Postoperative recovery is of importance if day surgery is to expand with all the advantages that this entails to patients and the health care system.

This study was designed to examine differences in recovery after propofol and thiopentone. The use of inhalational agents was avoided and all subjects were anaesthetised for dilatation and curettage. This minimised surgical differences between patients and ensured a small standard deviation in the mean duration of surgery. We believe these are essential requirements to improve the comparability of the study groups and to allow any differences to be attributed to the intravenous anaesthetic agents.

Recovery during the first few hours after intravenous anaesthesia was investigated widely.^{1,8-11} Most studies indicate that early recovery after propofol anaesthesia is more rapid than after thiopentone^{8,12-14} but there have been few reports of recovery after the patients have returned home.^{2,3}

The statistical analysis of the data in the two treatment groups was by Student's *t*-test and it was these differences that interested us most. Subsequently, all three groups were examined using analysis of variance (F tests). One-way analysis-of-variance F tests are designed to detect non-specific differences between the study groups, but are less sensitive than *t*-tests in detecting differences between two treatment groups. Thus the *t*-test may indicate a statistically significant difference between the treatment groups that is not supported by the F-test. The *t*-test, on occasions, failed to demonstrate a statistically significant difference between the groups, but F statistics showed a significant difference when the reference group was also considered. The implication of this is that there is a difference between one or both treatment groups and the reference group. For this reason, Student's *t*-test was used to compare each treatment group with the reference group.

Patients who received thiopentone were significantly more tired and drowsy, and less alert, than those anaesthetised with propofol 1 hour after surgery. No difference

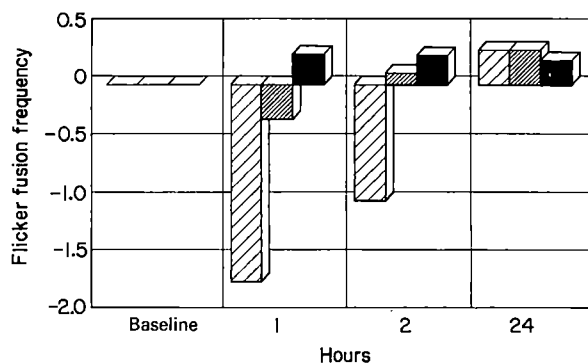


Fig. 1. Change in flicker fusion frequency. ▨, thiopentone; ▩, propofol; ■, control.

existed between the two groups at 2 hours, but at 24 hours the thiopentone group was again more tired and drowsy than the propofol group. F statistics failed to demonstrate the difference in feelings of tiredness at 24 hours, but confirmed the other differences. Patients in both treatment groups were significantly less alert than the reference group at pre-operative testing and remained so until the tests at 24 hours. This may reflect a high level of anxiety in the treatment groups in the pre-operative phase, which may also contribute to the differences seen after operation. Subjects are likely to be more relaxed when tested in their own homes at 24 hours. This pattern is similar to that noted for feelings of well-being recorded in a recent study.²

It appears from the 24-hour results for drowsiness and tiredness that propofol is beneficial. The scores in the thiopentone group returned virtually to normal, whilst those for propofol indicate that subjects were less tired and less drowsy than normal. A number of explanations are possible. Propofol produces euphoria in the postoperative period and the observed effect may be the result of sub-clinical euphoria. These patients may have been awaiting surgery for some time and this may reflect relief that the surgery is over. However, the relief may be masked in the thiopentone group by the hangover that follows thiopentone anaesthesia. A further explanation may be that this is a specific effect of propofol in reducing fatigue in the late postoperative period.

The results of the Bethune modification of Williams' memory function test showed a significant difference between the treatment groups in the error scores for new cards at 1 hour. There were no differences between treatment groups at any other time for new cards and the scores for the D card were never significantly different. However, examination of all three groups indicated a highly significant difference in the error scores for new cards presented at 1 and 2 hours. The score in the thiopentone group for the D card was significantly worse than in the reference group at 1 hour. The Bethune memory test examines memory for visually acquired information and the results imply that this is impaired postoperatively at both 1 and 2 hours. Thiopentone results in a greater impairment of memory at 1 hour than does propofol, which may be important if postoperative instructions are given before an early discharge.

The Wechsler Logical Memory Passages were used to examine the subjects' memory for auditory information. No significant differences were recorded between the treat-

ment groups. However, highly significant differences were observed at one hour and at 24 hours when the reference group was included in the analysis. Both study groups performed less well than the reference group at 1 hour. There was a significant difference between the groups at 24 hours that appears to result mainly from impairment of memory in the propofol group compared with the reference group; however, there was no significant difference in memory function between the propofol and thiopentone groups. This effect is difficult to explain. Propofol may have a specific inhibitory effect on auditory memory in the late postoperative period. Alternatively, this may be a reflection of the apparent reduction of tiredness and drowsiness in the propofol group in a situation where pressing domestic commitments may preoccupy the subjects whilst listening to the story passages. Such preoccupation is less likely to occur during performance of the other tests because they require input from the subject throughout the test. It should also be remembered that the reference group was included for comparison and is not a true control group.

The mean critical flicker fusion frequencies were examined in several ways. Initially, the mean frequencies were tested and there were no significant differences between the treatment groups. There was a significant difference ($p < 0.02$) in the results of the pre-operative tests when the reference group was included in this analysis. This may reflect an effect of anxiety in reducing critical flicker fusion frequency in the treatment groups, although the difference appears to be the result largely of a difference between the propofol and reference groups. The significance of the difference between the groups increased at one hour ($p = 0.0001$) and the difference remained significant at 2 hours ($p = 0.002$). The difference between both treatment groups and the reference group was highly significant. Only the difference between the propofol and reference groups was significant at 24 hours.

The sensitivity of critical flicker fusion frequency testing may be increased by subtracting each patient's pre-operative fusion frequency from the frequency at each test session. A significant difference was found between the treatment groups at one hour when the data were examined in this way. The differences were significant at both 1 and 2 hours, but not at 24 hours, when the reference group was included in this analysis.

Postoperative recovery is complex and it is unreasonable to assume that all neurological functions should recover at the same rate. There have been recent reports of apparent recovery from propofol anaesthesia followed by a further period of unconsciousness.¹⁵ Some of the inconsistencies observed in this study may reflect this type of process at a subclinical level, and the effect may be specific to specific neurological functions, e.g. auditory memory, and not confined to propofol anaesthesia. However, it is not surprising that such an effect should be observed with a drug that produces such good early recovery that a later reduction in conscious level is readily apparent. Further investigation in this area is required.

Appropriate anaesthetic techniques will assist the expansion of day-case surgical facilities in Britain. The results presented in this paper indicate that propofol is associated with superior subjective recovery from anaesthesia for up to 24 hours after surgery.

Acknowledgments

We gratefully acknowledge the assistance of Sister D.E. Sutherland and her day unit staff during this project and Dr L. Goldman for his advice on psychological testing.

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Platelet function after intramuscular diclofenac

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Summary

A randomised double-blind controlled study was performed to examine the effect of diclofenac on skin bleeding time and in vitro whole blood platelet aggregation. Twenty thoracotomy patients were studied; 10 were given diclofenac 75 mg intramuscularly at induction of anaesthesia, and 10 formed a control group. Skin bleeding times and platelet aggregation tests were performed the day before and repeated one hour after induction of anaesthesia. Diclofenac prolonged skin bleeding time and reduced platelet aggregation. There were no significant changes in the control group.

Key words

Analgesics; diclofenac.

Blood; coagulation.

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) to provide postoperative analgesia is becoming accepted practice. Intramuscular diclofenac was shown to be an effective analgesic,¹ but there is concern over the peri-operative use of the NSAIDs since they have potential side effects related to their mechanism of action, inhibition of the enzyme cyclo-oxygenase.² This enzyme is essential in platelets for the production of thromboxane A₂ which is an important mediator of platelet aggregation and vasoconstriction. These are processes that constitute the primary haemostatic response to vessel injury. Peri-operative administration of a NSAID could affect platelet function and inhibit haemostasis.

It is clear that NSAIDs and aspirin, which has a similar inhibitory effect on cyclo-oxygenase, inhibit aggregation and prolong bleeding time in volunteers,^{3,4} but there is little information on the peri-operative situation where the haemostatic response may be altered by the stress of surgery. It has recently become possible to study platelet aggregation in whole blood.^{5,6} This may be more physiological than traditional turbidometric methods using platelet-rich plasma, since the platelets are left in their natural *milieu* surrounded by red and white cells that can influence the aggregatory response.^{7,8} To our knowledge there is no previous study which uses the whole blood aggregation technique to investigate the effect of NSAIDs on platelet aggregation in the peri-operative situation. The aim of this study

was to investigate the effect of intramuscular diclofenac on skin bleeding time and whole blood platelet aggregation in a randomised, double-blind, controlled, peri-operative study.

Methods

Twenty ASA 1 and 2 patients aged 27 to 79 years undergoing thoracotomy were studied. The investigation was approved by the regional ethics committee and each subject provided written informed consent before enrolment. Exclusion criteria consisted of a history of peptic ulcer disease, bleeding tendency, asthma, allergies, recent aspirin or NSAID ingestion, and alcohol or narcotic abuse. The general anaesthetic technique was standardised (premedication, papaveretum and hyoscine; induction, thiopentone, suxamethonium; maintenance, nitrous oxide, oxygen, enflurane, fentanyl, alcuronium). Subcutaneous heparin (2500 IU subcutaneously, one hour before operation) was given as prophylaxis against venous thrombosis. All patients had intercostal nerve blocks before surgery (bupivacaine 0.5%). The subjects were randomly allocated so that 10 of them were given diclofenac 75 mg intramuscularly at induction of anaesthesia. The clinician who administered the injection was not otherwise involved in the study.

Skin bleeding times (all subjects) and platelet function

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Accepted 29 March 1990.

Table 1. Patient demography and blood loss. Values are shown as mean (SEM).

	Diclofenac	Control	p
Age; years	61.2 (4.4)	61.1 (4.1)	0.99
Height; cm	163.1 (1.4)	164.9 (1.7)	0.43
Weight; kg	62.5 (3.2)	67.7 (3.1)	0.25
Smoker; Y/N	4/6	2/8	0.63*
Sex; M/F	4/6	4/6	
Blood loss; ml	352 (125)	300 (165)	0.8

p, unpaired *t*-test; *Chi-squared test.

tests (10 subjects) were performed the day before surgery, and repeated one hour after induction of anaesthesia. Surgical blood loss was estimated by swab weight at operation.

Skin bleeding times were performed in duplicate on the forearm by the modified method of Ivy^{8,10} using a Simplate II bleeding-time device to standardise the length and depth of incision. Bleeding time was taken for the two incisions and the mean time recorded. The normal bleeding time is up to 10 minutes with this technique.¹⁰

Platelet aggregation was studied *in vitro* in five subjects in each group using a Clay-Adams Ultra-Flo whole blood platelet counter.^{6,11} Collagen (2 µg/ml, Semmelweis) was used as an aggregating agent. Whole venous blood was anticoagulated with 3.8% trisodium citrate. A red cell count was first determined from each sample and dialled into the whole blood platelet counter. A 10-µl aliquot was withdrawn and the baseline platelet count determined. The aggregating agent was then added and the sample stirred at a constant 1000 rpm and 37°C. Further aliquots were taken at 1, 3, and 5 minutes and the platelet count determined. Platelet aggregation was recorded as the percentage decrease in the platelet count from baseline to 1, 3, and 5 minutes.

Twenty patients entered and completed the trial. No adverse events were encountered.

The randomisation code was broken after the end of the study and the results obtained. Values are shown as mean (SEM). Subject demography and surgical blood loss were compared between the diclofenac and control groups using unpaired *t*-tests and Chi-squared tests. Before and after dose bleeding times and platelet aggregation were compared using paired *t*-tests. A *p* value of 0.05 or less was accepted as indicative of a significant difference, and all the tests were two-tailed. Ninety-five percent confidence intervals were calculated where appropriate (95% CI).

Results

There was no difference between the groups in age, height, weight, smoking history, or sex distribution (Table 1).

Table 2. Skin bleeding time. Bleeding times in seconds, mean (SEM), are shown for each group of 10 subjects before and one hour after induction of anaesthesia.

	Before induction	After induction	p
Diclofenac	250 (16.9)	330 (19.7)	0.0001
Control	226 (18.7)	222 (15.5)	0.66

p, paired *t*-test.

Surgical procedures were lobectomy (five diclofenac; four control), pneumonectomy (two diclofenac; two control), open lung biopsy (two in each group), pleurectomy (one diclofenac), and thoracotomy with no resection (two control). Blood loss did not differ between the groups (*p* = 0.80). There was a wide range of blood loss within each group (diclofenac 20 to 1200 ml; control 20 to 1700 ml).

Skin bleeding time (Table 2) was significantly prolonged one hour after diclofenac (*p* < 0.0001, 95% CI 47 to 115 seconds). There was no change in the control group (*p* = 0.66, 95% CI -24 to 16 seconds). The range of bleeding

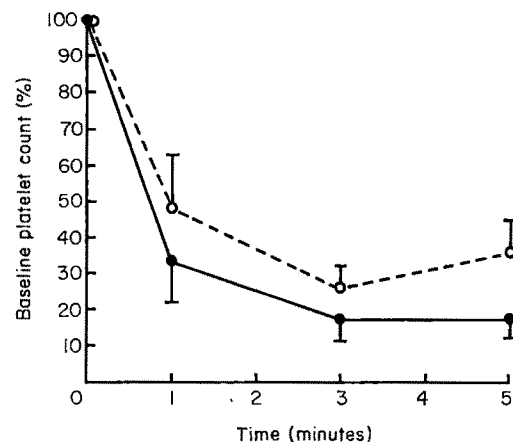
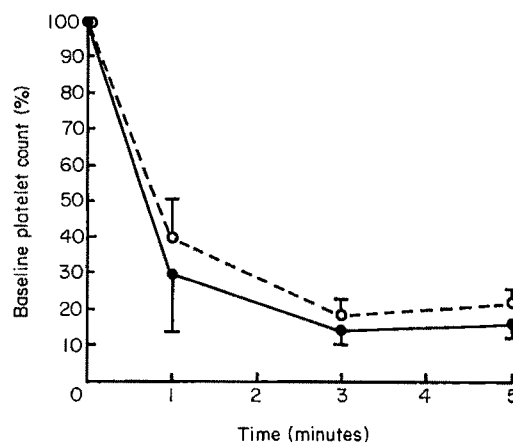
**Fig. 1.** Diclofenac group; five subjects. Collagen-induced platelet aggregation before (—●—) and one hour after (---○---) induction of anaesthesia. Mean (SEM).**Fig. 2.** Control group; five subjects. Collagen-induced platelet aggregation before (—●—) and one hour after (---○---) induction of anaesthesia. Mean (SEM).

Table 3. Diclofenac group platelet aggregation in five subjects. Values shown are the percentage decreases from baseline, mean (SEM), in platelet counts over 5 minutes in the presence of a collagen aggregator.

	Before induction	After induction	p
1 minute	66.4 (11.5)	51.8 (14.4)	0.077
3 minutes	82.6 (6.1)	73.8 (6.0)	0.024
5 minutes	82.6 (5.2)	64.0 (8.6)	0.038

p, paired *t*-test.

Table 4. Control group platelet aggregation in five subjects. Values shown are the percentage decreases from baseline, mean (SEM), in platelet counts over 5 minutes in the presence of a collagen aggregator.

	Before induction	After induction	p
1 minute	70.2 (15.8)	60.2 (11)	0.38
3 minutes	85.8 (3.2)	81.6 (4.4)	0.1
5 minutes	83.8 (3.8)	78 (4.3)	0.09

p, paired *t*-test.

times after diclofenac was from 247 to 480 seconds. The longest prolongation of bleeding time in a subject was from 350 seconds before diclofenac to 480 seconds after.

Figures 1 and 2 show the platelet counts in the 1-, 3-, and 5-minute sample for the diclofenac and control groups. Each figure shows the decrease in platelet count before and after injection of diclofenac (or control). Tables 3 and 4 show the change in platelet aggregation after diclofenac and the control. Diclofenac significantly reduced *in vitro* platelet aggregation at the 3- and 5-minute points, but not at one minute (Table 3). The 95% CI for the reduction in platelet aggregation with diclofenac at 5 minutes was from -35.5 to -1.7%. The control group showed a tendency towards reduced aggregation one hour after induction of anaesthesia (Fig. 2, Table 4), but this was not statistically significant (e.g. at five minutes $p = 0.09$, 95% CI -13 to 1.4%).

Discussion

This study has demonstrated that one hour after an intramuscular dose of diclofenac 75 mg skin bleeding time is prolonged and whole blood platelet aggregation is inhibited. Skin bleeding time is a useful clinical test of platelet function, and collagen-induced aggregation depends largely on thromboxane production by platelet cyclo-oxygenase.

Previous work found no change in bleeding time in patients 24 hours after an intramuscular dose of diclofenac 33.6 mg followed by an intravenous infusion of 6.7 mg/hour.¹² This was a surprising observation in view of the known effect of cyclo-oxygenase inhibitors on thromboxane production. Our work has shown that the clinically used dose of diclofenac 75 mg does affect skin bleeding time within one hour of intramuscular administration in patients having surgery.

The increase of bleeding time seen with diclofenac was statistically significant but modest (95% CI 47 to 115

seconds). The magnitude of this effect is similar to that seen with aspirin and may not be of clinical significance,³ since none of the bleeding times after diclofenac were above the normal upper limit of 600 seconds. Therefore, although diclofenac does affect platelets, it may not produce an abnormal haemostatic state in previously normal individuals. However, it was shown that certain members of the population are more sensitive to the haemostatic effects of aspirin than others¹⁰ and so some patients could have a greater effect from diclofenac than seen in this study.

The tendency towards reduced platelet aggregation in the control group was statistically insignificant, but since it could reflect the effects of anaesthesia and surgery on platelet function it may merit further study.

The effect of intramuscular diclofenac 75 mg on surgical blood loss is unclear. This investigation was not designed to address that problem. Previous studies have found that pre-operative administration of lower doses of diclofenac do not increase blood loss after gynaecological laparotomy (33.6 mg intravenously)¹² or transurethral prostatectomy (50 mg orally).¹³ Experience with other NSAIDs suggests that the dose used is important in determining whether blood loss is increased or not. For example, in gynaecological laparotomy pre-operative intravenous indomethacin 0.8 mg/kg increased intra-operative blood loss,¹⁴ but 25 mg did not.¹² The clinically used intramuscular dose of diclofenac is 75 mg. Further studies are required to ascertain whether the platelet dysfunction produced by diclofenac 75 mg given intra-operatively is associated with increased blood loss.

In conclusion, diclofenac 75 mg intramuscularly does impair platelet function within one hour. This should be considered if blood loss seems excessive when diclofenac has been given intra-operatively. In addition it may be prudent to avoid the use of diclofenac or another NSAID in the presence of other defects of haemostasis or coagulation. Indeed, the administration of a NSAID could reveal a subclinical haemostatic problem, and if this was suspected the skin bleeding time would be a useful clinical test to perform.¹⁵

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Posture and epidural catheter insertion

The relationship between skill, experience and maternal posture on the outcome of epidural catheter insertion

P. A. STONE, A. W. A. KILPATRICK AND J. THORBURN

Summary

This study was undertaken to investigate the outcome of epidural catheter insertion in the sitting or lateral position in mothers during labour. An initial prospective randomised study period (144 patients) suggested that the sitting position offered some superiority over the lateral in terms of technical ease of insertion. It was concluded, by minimising the subjective aspects in a follow-up, prospective nonrandomised study period (152 patients), that the determining factor lies in the skill and experience of the anaesthetist. There was no significant difference in complication rates or maternal discomfort between the two positions in either study period.

Key words

Anaesthetic techniques, regional; epidural. Complications.

The insertion of epidural catheters for analgesia in labour may be carried out with the patient in the sitting or lateral position. The position used is primarily related to the preference of the anaesthetist and is based on the technique taught during the anaesthetist's initial training period. No previous studies have been undertaken to evaluate the effect of position with respect to successful identification of the epidural space. The aim of this study was to investigate the effect of maternal posture on the ease of insertion of epidural catheters for analgesia in labour, the associated complication rates and patient discomfort.

Methods

To minimise the subjective aspects of the study it was divided into two sections. In the first study period the position of the patient was randomly allocated, and in the second the anaesthetist used the position that he (she) considered appropriate.

Randomised study period. One hundred and fifty patients who had requested epidural analgesia during labour consented to take part in the study. They were randomly allocated to have the epidural sited in the sitting or lateral position. All epidurals were performed by anaesthetists who had at least completed their training period in obstet-

ric anaesthesia and were happy to perform the technique in either position.

Nonrandomised study period. Anaesthetists were asked to perform epidurals with the patient in the position which they considered to be most appropriate, to record details of the position used, whether it was chosen as the anaesthetist's preference, patient's preference or for any other reason. The anaesthetist's usual preference, if any, was stated and any reason for deviation from this. One hundred and fifty two patients were studied in this manner.

The methods were as outlined below during the two study periods. At least 16 anaesthetists, from registrar to consultant grade, participated in the study. A standardised epidural technique was employed, using loss-of-resistance via a midline approach. On identification of the epidural space, 5 ml of 'solution' (normal saline or local anaesthetic) were injected via the needle before an attempt was made to pass the epidural catheter 5 cm into the epidural space. The needle was then removed and the catheter withdrawn to the desired position. Tuohy needles 16- or 18- gauge were used (Portex Minipack systems); size was chosen according to the anaesthetist's preference.

The anaesthetist who performed the epidural insertion recorded details as follows: patient position; patient's height and weight; gauge of Tuohy needle; time taken from

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Accepted 4 April 1990.

cleaning patient's back until injection of test dose via the epidural catheter; the insertion time; ease of identification of the epidural space, by a 10-cm visual analogue scale; number of skin punctures with Tuohy needle; reasons for more than one skin puncture; whether the catheter passed freely 5 cm into the epidural space; any problems encountered; complications in relation to siting of the epidural catheter; was the patient moved to the alternative position, and, if so, why?

The patient was asked, once analgesia was established, to indicate the overall discomfort associated with the procedure by marking a 10-cm visual analogue scale. In addition, she was asked to indicate which position she would prefer, if any, if she were ever to have a further epidural.

Analysis of results was by Chi-squared, Mann-Whitney *U* tests and Spearman's correlation as appropriate. A probability value of less than 0.05 was considered to indicate statistical significance.

Results

Randomised study period (Table 1)

After enrolment of 150 patients into the study sufficient data were available for analysis in 144 patients. The patients were comparable with respect to their body mass indices (i.e. weight/height²). Sixteen-gauge needles were more commonly used in both the sitting and lateral groups; comparable percentages of each group were carried out with this size.

The mean insertion time was significantly longer in the lateral group. Failure to pass the catheter freely which led to more than one skin puncture was also more common in this position. More patients in the lateral group expressed a preference for the other position if they were to have another epidural. The need to move patients to the other position was more frequent in those allocated to the lateral position.

The difficulty scores, use of more than one skin puncture, individual complication rates and maternal pain scores were similar for each maternal position. There was evidence of blood vessel puncture in 18% of patients who were in the sitting position compared with 12% who were in the lateral; this difference is not statistically significant. There were two inadvertent dural punctures with the needle in the lateral group, and one with the catheter in each group.

There was a positive correlation between body mass index and epidural insertion time in the lateral group, with a Spearman correlation coefficient of +0.41 ($p < 0.01$). No significant correlation was found between body mass index and epidural insertion time in the sitting group.

The mean time for epidural insertion in patients with a body mass index value that exceeded 30, that is the relatively obese patients, was significantly longer in the lateral group ($n = 12$), in whom the mean (SD) insertion time was 17.01 (15.91) minutes compared with 6.54 (3.39) minutes in the sitting group ($n = 18$); $p < 0.01$; Mann-Whitney *U* test).

Nonrandomised study period (Table 2)

The patients were comparable with respect to their body mass indices. Sixteen-gauge Tuohy needles were again used more often; there was no difference in the incidence of use between those in the sitting and lateral positions. The time for insertion, difficulty scores, failure to pass the catheter freely and need for more than one skin puncture were similar for each maternal position. There were no inadvertent dural punctures in either group, and evidence of blood vessel puncture was more common in the sitting group (8%) than the lateral group (4%) although this difference is not statistically significant.

Maternal pain scores and position preference were similar for each group, as was the need to move patients to the other position. There was positive correlation in the sitting position between body mass index and time taken

Table 1. Randomised study results.

	Sitting ($n = 71$)	Lateral ($n = 73$)	
Body mass index (SD)	27.8 (4.3)	26.4 (3.8)	ns
16-G needle size, when answered, (others 18-G)	45/68 = 66%	44/71 = 62%	ns
<i>Ease of insertion</i>			
Mean time for insertion; minutes (SD)	6.56 (4.57)	10.9 (9.91)	$p < 0.05$
Mean difficulty score (SD) (visual analogue scale)	1.79 (2.11)	2.44 (3.01)	
Failure to pass catheter freely	13/71 = 18%	17/67 = 25%	ns
Failure to pass catheter freely, leading to more than one skin puncture	0/71 = 0%	9/67 = 13%	$p < 0.05$
More than one skin puncture	17/70 = 24%	25/72 = 35%	ns
<i>Complications</i>			
Evidence of blood vessel puncture	13/71 = 18%	9/73 = 12%	ns
Dural tap, needle	0/71 = 0%	2/73 = 3%	ns
Dural tap, catheter	1/71 = 1%	1/73 = 1%	ns
<i>Maternal comfort</i>			
Mean maternal pain score (SD)	1.97 (1.66)	1.69 (1.64)	
Pain score > 5.5	6%	5%	
< 3.5	76%	79%	
Maternal position preference, for other position	4%	15%	$p < 0.05$
Need to move patient to other position	1	17%	$p < 0.01$

ns, not significant.

Table 2. Nonrandomised study results.

	Sitting (n = 96)	Lateral (n = 56)	
Body mass index (SD)	27.38 (4.54)	27.17 (3.72)	ns
16-G needle used (18-G in others)	83%	37/55 = 67%	ns
<i>Ease of insertion</i>			
Mean time for insertion; minutes (SD)	6.28 (3.2)	5.37 (4.22)	ns
Mean difficulty score (SD) (visual analogue scale)	1.45 (1.85)	1.35 (1.94)	ns
Failure to pass catheter freely	13%	5/46 = 11%	ns
Failure to pass catheter freely, leading to more than one skin puncture	2%	1/46 = 2%	ns
More than one skin puncture	9%	8/56 = 14%	ns
<i>Complications</i>			
Evidence of blood vessel puncture	8%	2/96 = 4%	ns
Dural tap, needle	0%	0/56 = 0%	
Dural tap, catheter	0%	0/56 = 0%	
<i>Maternal comfort</i>			
Mean maternal pain score (SD)	2.11 (1.85)	1.81 (1.55)	
Pain score > 5.5	4%	2%	
< 3.5	76%	75%	
Maternal position preference, for other position	7%	4%	ns
Need to move patient to other position	1%	0%	ns

ns, not significant.

for insertion of the epidural, with a Spearman correlation coefficient of +0.45 ($p < 0.001$). No significant correlation was found in the lateral group.

In the relatively obese patients with a body mass index value that exceeded 30, the time taken for insertion was similar in the two positions. The mean insertion time (SD) was 6.19 (2.91) minutes in the lateral group and 8.84 (4.20) minutes in the sitting group. This difference is not statistically significant (Mann-Whitney *U* test).

The insertion times were analysed in relation to the anaesthetist's usual maternal position preference. In the lateral position, if that was not the usual position preference ($n = 31$), mean time for insertion was significantly longer than if the usual preference was lateral ($n = 25$). The mean (SD) insertion times were 7.1 (4.7) and 3.2 (2.2) minutes respectively, with a p value < 0.01 (2-tail Mann-Whitney *U* test). However, in the sitting position, if that was not the usual position preference ($n = 22$), the mean (SD) time for insertion of 6.2 (3.9) minutes was similar to that of 6.3 (3.0) minutes when the usual preference was sitting ($n = 72$). This difference is not statistically significant.

Discussion

The patient position used for insertion of epidural catheters for analgesia in labour is generally related to the personal preference of the anaesthetist. Fetal condition or maternal factors rarely influence the choice of position. The preference of the anaesthetist is likely to be the position in which he (she) was taught to perform epidurals. Obstetric anaesthetic texts differ in position favoured. Bonica¹ points out that the lateral position is more comfortable and safer for the patient, and suggests that in the sitting position there is risk of syncope and that increased cerebrospinal fluid pressure may increase the chance of inadvertent dural puncture. Marx and Bassell² cite possible advantages of the sitting position as ease of maintenance of position, central position of the lumbar spines and that the patient may find

the lateral position awkward. Moir and Thorburn³ suggest that greater spinal flexion may sometimes be achieved in the sitting position. Crawford⁴ discusses the lateral position only in his text on obstetric anaesthesia. There are no previous reported studies which have attempted to investigate the effects of maternal position on the ease of insertion of epidural catheters, complications and maternal comfort during insertion.

Patient position was randomised in the initial study period, and as blinding is clearly not possible in such a study, subjective aspects related to personal preference and experience may have influenced outcome. It should be stressed that anaesthetists who performed the epidurals in the study all considered themselves competent to use either position.

The objective measure of time for insertion, using randomised positions, was significantly longer in the lateral group of patients and more patients were moved from the lateral to sitting position to secure epidural insertion. There was increased difficulty in passing the catheter in the lateral patients, but no significant difference in complication rates. It was also noted that the relative heaviness of the patient, as indicated by body mass index, correlated with time for insertion in the lateral group only. These results suggest that the sitting position is superior to the lateral position.

The second study period was carried out to attempt to identify the effects of personal preference and experience on the relationship between maternal position and insertion of epidural catheters. In this part of the study the time for insertion was comparable in the two groups. There were no significant differences in any of the recorded technical difficulties, complications or maternal comfort considerations.

The epidural insertion time was analysed in relation to the usual position preference of the anaesthetist. It is of interest to note that when the lateral position was used, and it was not the usual preference of the anaesthetist, this was associated with increased time for insertion. Personal preference did not appear to influence the time taken in the

sitting position, however. It seems, therefore, that the anaesthetist who prefers the lateral position may have less difficulty adapting to the other position than the anaesthetist who prefers to use the sitting position.

The overall incidence of evidence of blood-vessel puncture was 10.8%, which is of interest since in a previous study in the authors' department⁵ the incidence was 18%. Then, 16- and 18-gauge needles were studied, patient position was not accounted for and there was no standardisation of injection of fluid via the needle before an attempt was made to pass the catheter. Verniquet's study⁶ of blood-vessel puncture, which demonstrated the beneficial effect of injection of fluid via the needle, was carried out with patients in the lateral position. The increased incidence of evidence of blood vessel puncture in the sitting groups of patients in our study did not achieve statistical significance, but it would seem likely that in the sitting patient, increased epidural venous pressure would increase the incidence of vessel puncture. However, unaccustomed use of the lateral position is likely to lead to difficulty in identification of the midline, rendering a higher risk of blood-vessel puncture than when the anaesthetist is experienced in, and prefers, the lateral position.

Anaesthetists who have their initial obstetric training at the Queen Mother's Hospital are taught to administer epidural anaesthesia with the patient in the sitting position. Not all anaesthetists involved in the study were trained in the Queen Mother's Hospital, but the majority prefer to insert the epidural in the sitting position. From the first part of the study it is clear that the lateral position proved more difficult despite the anaesthetists being happy to employ that position. The nonrandomised part of the study illustrates the fact that where the anaesthetist is allowed to

choose the preferred position the posture of the patient does not affect the efficacy of administration of the block. Complications and maternal comfort during epidural insertion were not significantly affected by posture in either study period.

It is clear, therefore, with respect to maternal posture and ease of insertion of epidural catheters that the determining factor is the skill and experience of the anaesthetists and these results suggest that the anaesthetist who is already trained should be permitted the freedom of choice of maternal position and not be encouraged to adopt a less familiar approach.

Acknowledgments

The authors thank the anaesthetic staff at the Queen Mother's Hospital for taking part in this study, and in particular Dr C.S. Cairns (Consultant Anaesthetist) for his support in establishing the study.

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The laryngeal mask airway in paediatric anaesthesia

D. F. JOHNSTON, S. R. WRIGLEY, P. J. ROBB AND H. E. JONES

Summary

Forty-eight children, aged between 2 and 10 years, admitted as day cases for otological surgery were allocated at random into two groups. The first group was anaesthetised using a standard facemask, and the second with a laryngeal mask airway. The laryngeal airway produced a satisfactory airway in all children, and was inserted on the first attempt in 67% of patients. Hypoxia was significantly less frequent in the laryngeal airway group ($p < 0.05$), and there were significantly fewer interruptions to surgery than in the facemask group ($p < 0.001$). Patient safety, operating and anaesthetic conditions were all considered superior in the laryngeal airway group.

Key words

*Equipment; laryngeal mask airway.
Surgery; paediatric, otological.*

The laryngeal mask airway was introduced recently into anaesthetic practice. It provides an alternative to the facemask or tracheal tube for airway maintenance during general anaesthesia with spontaneous breathing. The Brain laryngeal airway¹⁻³ comprises an inflatable mould that rests above the larynx to provide a seal without the need for tracheal intubation. It was shown to be easy to introduce in adults by anaesthetists with no previous experience of its use, and provides a clear airway with the added advantage of freeing the anaesthetists' hands for other duties.⁴

It was shown in children, that tracheal intubation can be associated with postoperative discomfort and subtle changes in laryngeal function.⁵ Our recent work shows there is less risk of laryngeal injury and oedema after use of the laryngeal airway compared with a tracheal tube for airway maintenance.⁶ The majority of paediatric day-case otological procedures in our hospital are short microsurgical operations. Tracheal intubation is believed to be relatively contraindicated in this group of children because of the possible risks of laryngeal oedema and trauma. However, a facemask may necessitate interrupting the procedure from time to time, because airway maintenance may be difficult as a result of the proximity of the surgical field to the airway. These may lead to the occurrence of airway obstruction with a subsequent decrease in depth of anaesthesia, hypoxia, and patient movement. The latter is poten-

tially hazardous in microsurgical otological procedures, no matter how minor.

The laryngeal mask airway appears to be a suitable alternative method for the maintenance of an airway during this type of day-case otological surgery, and one which avoids some of the risks inherent with using either a facemask or tracheal tube, with the added advantage of a stationary microsurgical field.

Method

The study was approved by the Hospital Ethics Committee and informed verbal consent was obtained from the parent or adult accompanying the child.

Forty-eight children, between the ages of 2 and 10 years, of ASA 1 or 2, who presented for day-case otological surgery, were entered into the study. The children were assessed in the usual way before surgery and the normal exclusion criteria for day-case surgery applied. The type of surgery was limited to: examination of ears under anaesthesia; myringotomies with or without the insertion of grommets or Goodes tubes. These were considered to be comparable procedures. In all children, both ears were examined.

All children had EMLA cream (Astra) applied to the

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Accepted 1 April 1990.

dorsum of either hand at least one hour before operation, but no other premedication was given.

Indirect arterial blood pressure, electrocardiogram, and oxygen saturation were monitored in all children. Data were recorded at 2-minute intervals, starting before induction of anaesthesia. Anaesthesia was induced in theatre, after establishment of intravenous access, with sodium thiopentone (4–6 mg/kg) in the presence of the parents and ward nurse. Increments of sodium thiopentone were given as necessary. Anaesthesia was maintained with isoflurane 1–4% in oxygen (4 litres/minute) and nitrous oxide (6 litres/minute). Vaporizer settings were recorded at 2-minute intervals with the other measurements. An Ayre's T-piece with reservoir bag was used for children of up to 20 kg body weight, and a Bain's system for those over 20 kg. Intramuscular codeine phosphate (1 mg/kg) was administered to all children during operation.

Once the depth of anaesthesia was considered satisfactory for either surgery to proceed with a facemask, or for the laryngeal airway to be inserted, the children were allocated to one of the two groups at random.

In the first group of children (FM) anaesthesia was maintained with the facemask, and in the second group (BLA) a laryngeal airway of the appropriate size was inserted. The ease of insertion, and number of attempts required were recorded. The isoflurane was switched off when surgery was completed and 100% oxygen administered. In the BLA group the laryngeal airway was removed by the anaesthetist in theatre. Both groups then received oxygen via an MC mask while in transit to the recovery room and until awake. All surgical procedures were performed and anaesthetics given by the authors. The data were recorded by an independent trained observer. The following times were recorded: induction of anaesthesia; randomisation; start of surgery; end of surgery; transfer to recovery; and wake up time.

Patient comfort was assessed where possible, before and after surgery, by asking the child whether they had a sore throat. The throat was also examined for pharyngeal oedema, erythema, or bruising by one of the surgical investigators after surgery. The anaesthetic and surgical conditions were assessed using a scoring system related to anaesthetic events, and surgical field as perceived by the surgeon. These are summarised in Table 1. The study could not be blinded in technique to the surgeon. The data collected were analysed using Chi-squared and Student's *t*-test where appropriate.

Table 1. Criteria used to score anaesthetic and surgical conditions.

Anaesthetic score
A Uneventful procedure
B Oxygen saturation less than or equal to 94%
C Patient moving (\pm B)
D Coughing or laryngospasm (\pm B or C)
Surgical score
A Completely ideal operating field
B Minor movements of surgical field
C Rearrangement of airway not disrupting surgery
D Rearrangement of airway: surgery stopped < 30 seconds
E Rearrangement of airway: surgery stopped > 30 seconds

Results

The two groups were comparable for age, weight, sex, and ethnic origin (Table 2). In the FM group 21 patients were ASA 1 and three ASA 2, two of whom suffered from Down's syndrome, with no serious cardiac anomalies or obvious airway problems. The third child suffered from asthma that required treatment with inhaled bronchodilators. In the BLA group all were ASA 1.

The mean heart rate, arterial blood pressure (systolic and diastolic), oxygen saturation, total dose of thiopentone, and percentage of isoflurane delivered are shown in Table 3. The mean length of surgical time in the BLA group (5.77 minutes) was significantly shorter ($p < 0.05$) than in the FM group (7.75 minutes), Table 4. Tables 5 and 6 show the anaesthetic and surgical conditions with the number of children in each group. It can be seen that in the BLA group there is only one child in whom the surgical field was less than ideal. This was due to the child coughing. In the FM group, it was often necessary to interrupt surgery in order to rearrange the airway, or for a tremor to be

Table 2. Demographic data of children in both groups. Values expressed as mean (SD) range or total numbers.

	Laryngeal airway	Facemask
Age; years	6.74 (2.67) 2.75–10.67	6.69 (1.98) 2.75–10.75
Weight; kg	22.81 (8.04) 13–45	23.20 (7.70) 14–37
Female	12	11
Male	12	13
ASA 1	24	21
ASA 2	0	3
Caucasian	22	18
Negroid	2	4
Mixed	0	2

Table 3. Measured physiological variables and drugs given. Values expressed as mean (SD).

	Laryngeal airway	Facemask
Heart rate; beats/minute	120.6 (14.6)	116.7 (15.7)
Systolic blood pressure; mmHg	114.6 (10.3)	112.2 (12.9)
Diastolic blood pressure; mmHg	61.6 (8.8)	60.0 (10.0)
Percentage oxygen saturation	97.3 (1.9)	96.5 (1.7)
Percentage isoflurane used	3.0 (0.5)	2.8 (0.5)
Total dose thiopentone; mg/kg	6.8 (1.4)	7.2 (2.3)

Table 4. Measured and derived times mean (SD).

	Laryngeal airway	Facemask
Randomisation; minutes	3.82 (1.52)	3.84 (1.54)
Start of surgery; minutes	5.82 (2.20)	4.84 (1.89)
End of surgery; minutes	11.59 (2.20)	12.59 (3.78)
Time awake; minutes	31.21 (7.52)	33.24 (9.59)
Duration of surgery; minutes	5.77 (1.75)	7.75 (4.28)*

* $p < 0.05$ (Student's *t*-test).

The laryngeal airway appears to have both surgical and anaesthetic advantages in this type of paediatric surgery. The laryngeal mask airway produces a more secure and safe airway than the facemask, as shown by the different incidence of episodes of desaturation in the BLA group (2) compared with the FM group (14) after randomisation. This may be partly explained by the close proximity of the surgical field to the airway that makes it difficult to main-

tain the airway with a facemask. The surgical score was probably a direct result of a more secure airway that led to less interruption, and a motionless surgical field with the laryngeal airway.

The duration of the operating time was between 6 and 8 minutes in this study, with experienced surgeons; during earlier training, surgery can take up to 25–30 minutes. It would seem, in these circumstances, that the laryngeal mask would be ideal and would give a secure airway with a still surgical field to aid teaching and ensure patient safety.

The aim of this study was to assess the use of the laryngeal mask airway in paediatric otological surgery. Two of the patients presenting had Down's syndrome, in whom pre-operative assessment did not indicate a potential airway problem; both were in the FM group. One had an uneventful anaesthetic and the other two episodes of low oxygen saturation. Children with congenital abnormalities often have potentially difficult airways and associated middle-ear problems, e.g. Pierre-Robin and Treacher-Collins syndromes. The role of the laryngeal mask in these situations still needs to be evaluated although there are isolated case reports⁷ of the successful use of the laryngeal mask in children with some anomalies.

The Brain laryngeal airway can be safely and effectively used in paediatric patients and appears to have advantages in comparison with the facemask for otological surgery.

Acknowledgments

We are grateful to the nursing staff and operating department assistants in the Day Case Theatre at Guy's Hospital for their help and cooperation with this study. We also thank Dr J.E. Fairfield and Professor R.M. Jones for their assistance in the preparation of this paper.

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Volume therapy with hypertonic saline hydroxyethyl starch solution in cardiac surgery

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Summary

The ideal solution for volume therapy remains controversial. In cardiac surgery, haemodynamic efficacy as well as the influence of extracorporeal oxygenation are of major interest when administering volume. The present study examines the effects of a new hypertonic saline hydroxyethyl starch solution in comparison to a 6% hydroxyethyl starch solution on haemodynamics and laboratory variables. Patients scheduled for elective aortocoronary bypass grafting received hypertonic saline hydroxyethyl starch ($n = 10$) or hydroxyethyl starch ($n = 10$) after induction of anaesthesia in order to double baseline pulmonary capillary wedge pressure. Ten patients without volume therapy served as a control group. Significantly less hypertonic solution than standard solution was effective in doubling pulmonary capillary wedge pressure. Fluid requirements in the patients who received the hypertonic solution were significantly less during, as well as after, cardiopulmonary bypass in comparison to those in the other groups. Cardiac index increased most in the patients who received the hypertonic solution (+34.8%), as did right ventricular end-diastolic volume. Patients in that group showed the highest decrease in total systemic resistance (−29.8%), whereas arterial pressure and right ventricular ejection fraction remained almost unchanged in all groups. No negative alteration in coagulation or organ function was demonstrated within the investigation period. It can be concluded that hypertonic saline hydroxyethyl starch solution seems to be a valuable alternative to conventional volume therapy in cardiac surgery.

Key words

Blood; replacement, hydroxyethyl starch, hypertonic saline. Surgery; cardiac.

Absolute or relative blood volume deficits occur often during cardiac surgery.¹ Pre-operative condition, pre-operative medication and anaesthesia may significantly alter the patient's blood or plasma volume, and bleeding may decrease the patient's blood volume within the operation period. The use of plasma substitutes is often necessary. The choice between crystalloids and colloids for volume replacement remains controversial.^{2–4}

The use of hypertonic saline (HS) solutions was advocated for resuscitation from shock induced by haemorrhage or thermal injury.^{5–11} Concentrations of saline in these studies ranged from 3% to 7.5% and the term 'small volume resuscitation' was used because HS was much more effective than resuscitation with an equal volume of physiological saline solution.⁹ The haemodynamic response of HS may be transient^{12–14} and dextran may be added to it to prolong its haemodynamic efficiency. However, dextran is sometimes associated with severe anaphylactic reactions and alterations in coagulation.^{17,18}

Hypertonic fluids may also be of interest for plasma volume expansion in cardiac surgery, so we investigated the haemodynamic and laboratory effects of a newly developed combination of hypertonic sodium chloride and hydroxyethyl starch (HES) solution in a clinical setting.

Methods

Patients

Thirty patients scheduled for elective aortocoronary bypass grafting were investigated. Informed consent to participate in the study was obtained from each patient according to the protocol of the Human Ethics Committee of the hospital.

Inclusion criteria were normal left ventricular function (ejection fraction > 50%; left ventricular end-diastolic pressure < 20 mmHg), absence of pre-operative coagulation disorders, arrhythmia and concomitant valvular

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Accepted 2 April 1990.

disease, and a pulmonary capillary wedge pressure (PCWP) below 4 mmHg. None of the patients was taking cardiac glycoside, beta-blocker, or antiarrhythmic therapy. The patients were allocated at random to one of three groups. Patients in group 1 (HS-HES) received a new hypertonic solution prepared in hydroxyethyl starch solution (72 g/litre NaCl, 60 g/litre HES 200/0.5, osmolarity 2400 mosmol/litre) via a central line in order to double baseline PCWP within 20 minutes. Those in group 2 (HES) received a standard hydroxyethyl starch solution (6% HES 200/0.5, 9.0 g/litre NaCl; osmolarity 309 mosmol/litre) in order to double baseline PCWP within 20 minutes. Patients in group 3 ($n = 10$) received no volume replacement. Surgery started when volume replacement in groups 1 and 2 was complete.

Anaesthesia and cardiopulmonary bypass (CPB)

All patients were premedicated with morphine (0.15 mg/kg) and flunitrazepam (0.02 mg/kg) 1 hour before admission to the operation room. Induction (fentanyl 5 µg/kg; midazolam 0.1 mg/kg; pancuronium bromide 0.1 mg/kg) and maintenance of anaesthesia (fentanyl total dose 35 µg/kg; midazolam total dose 0.7 mg/kg; pancuronium bromide total dose 0.25 mg/kg) were performed by intravenous bolus injections and were identical in all patients. The lungs were ventilated mechanically with an F_{IO_2} of 1.0 before and after CPB. F_{IO_2} was adjusted according to blood gas analysis in the intensive care unit (ICU).

Anticoagulation before CPB was achieved by administration of 300 IU/kg heparin. Cardiopulmonary bypass was performed with a membrane oxygenator, and a flow of 2.4 (litres/minute)/sq m was maintained under almost normothermic conditions (lowest mean (SD) rectal temperature 34.2 (0.5)°C). Priming of the extracorporeal system consisted of crystalloids only (2200 ml); Ringer's solution was added as needed to maintain flow and packed red cells were added to the perfusate if the haemoglobin concentration dropped below 7 g/dlitre. A mono-atrial cannula was used to return venous blood to the system, and the operation was carried out in 'partial' bypass. The perfusate was concentrated within 20 minutes after the start of CPB, using a centrifugation method ('cell saver III', Hemonetics, Munich—2 cycles standardised). The fluid balance during CPB was calculated from volume application during CPB, urine output, and blood loss. The cell saving system was used to concentrate the blood that remained in the extracorporeal system after termination of CPB, and the concentrated blood ('washed red cells') was retransfused until the end of the operation.

Measured variables

Heart rate, mean arterial pressure (MAP), pulmonary arterial pressure (PAP), pulmonary capillary wedge pressure (PCWP), cardiac output (CO, thermodilution technique), right atrial pressure (RAP), and right ventricular systolic pressure (RVPSyst) were recorded, and derived variables (cardiac index (CI) total systemic resistance (TSR)) were calculated from standard formulae. The pulmonary artery catheter inserted via the right jugular vein was intended specifically to measure right ventricular variables. It is equipped with a fast response thermistor (FRT) and two electrodes for intracardiac electrocardiographic (ECG) recording. This thermistor allows recording

of plateaux of the rapid step changes on the descending limb of the typical thermodilution washout curve. Using a bedside microprocessor (REF-1, Edwards Lab., St Ana, CA), repetitive determinations of right ventricular ejection fraction (RVEF), right ventricular end-systolic volume (RVESV), and right ventricular end-diastolic volume (RVEDV) are possible. Detailed techniques of this monitoring instrument were reported elsewhere¹⁹ and accuracy as well as validity were documented in comparison to standard techniques for assessing right ventricular function.^{20,21}

In addition to haemodynamic values, haemoglobin (Hb), plasma haemoglobin (free Hb), red cell volume (RCV), fibrinogen, AT-III, partial thrombin time (PTT), thrombin time (TT), prothrombin time (PT), number of platelets, colloid osmotic pressure (COP; Sartorius membrane 20 000 dalton), osmolarity (blood and urine), and blood viscosity were measured before, during and after CPB as well as on ICU until the morning of the first postoperative day. Free-water clearance (CH_2O) was calculated in order to estimate renal function. The need for volume application (packed red cells when Hb was below 9 g/dlitre; Ringer's solution when PCWP was below 7 mmHg), and the use of catecholamines were determined by anaesthesiologists not involved in the study.

Haemodynamic and laboratory measurements were performed: during steady-state anaesthesia, in stable haemodynamic conditions (baseline values); at the end of volume application (after doubling PCWP); 20 minutes after finishing volume application; 40 minutes after finishing volume application (before start of CPB); 20 minutes after start of CPB (laboratory values only); immediately after termination of CPB; at the end of the operation; 5 hours after termination of CPB (on ICU, laboratory values only); and on the morning of the first postoperative day (laboratory values).

Statistics

Statistical analysis was carried out with 1- and 2-factorial analyses of variance with Scheffe testing and with analysis of covariance. Differences were considered significant when p was < 0.05 .

Results

There were no significant differences among the groups with regard to biometric data, pre-operative myocardial function, and duration of cardiopulmonary bypass (Table 1).

Significantly less HS-HES solution (4.5 (0.5) ml/kg) was necessary to double baseline PCWP in comparison to 6% HES solution (10.1 (1.4) ml/kg). PCWP never exceeded 10 mmHg. Fluid balance during CPB was negative in the HS-HES patients (-0.03 (0.01) (ml/kg)/minute), whereas HES patients ($+0.06$ (0.02) (ml/kg)/minute) and the control patients ($+0.13$ (0.03) (ml/kg)/minute) had a positive fluid balance (Table 1). Postoperatively, the HS-HES group required significantly less volume (400 (110) ml) than the HS patients (880 (200) ml) or the control group (1290 (200) ml).

There were no differences in MAP or HR among the groups within the entire investigation period (Table 2). HR increased slightly until the start of CPB. Elevation of

Table 1. Details of the patients, their pre-operative heart function, data from cardiopulmonary bypass (CPB), and postoperative variables. Data are presented as mean (SD).

	HS-HES	HES 6%	Control
Age; years	59.9(6.6)	61.6(5.0)	59.4(4.4)
Height; cm	169.9(6.6)	171.6(5.3)	169.9(10.4)
Weight; kg	74.1(9.2)	77.9(8.6)	73.8(10.8)
LVEF; %	69.2(12.2)	70.9(11.0)	69.3(7.5)
LVEDP; mmHg	12.0(2.7)	11.3(5.0)	14.9(9.9)
CPB; minutes	75.8(18.2)	73.6(16.8)	66.1(12.5)
Ischaemia; minutes	50.2(12.2)	45.2(11.1)	40.9(7.2)
Fluid balance during CPB; (ml/kg)/minute	-0.03(0.01)**	+0.06(0.02)	+0.13(0.02)
Blood loss; ml			
Operation day	125(77)	196(143)	192(109)
First postoperative day	361(122)	538(123)	585(199)
Volume of Ringer's solution; ml	400(110)**	880(200)	1290(210)

LVEF, left ventricular ejection fraction; LVEDP, left ventricular end-diastolic pressure.

**p < 0.05 compared to HES and control groups.

PCWP was maintained in both volume groups until 40 minutes after termination of volume administration. Cardiac index (CI) increased significantly more in the HS-HES group (+34.8%) than in the HES patients (+24.4%), and decreased in the control patients (-14.2%). TSR decreased significantly in the HS-HES (-29.6%) and the HES patients (-11.2%), but increased in the control patients (+17.9%) (Fig. 1). Blood viscosity decreased most in the HS-HES patients (Fig. 1), but no correlation between the degree of decrease in TSR and blood viscosity could be demonstrated (analysis of covariance). RVPsyst increased significantly in both volume groups; RVEDP and RAP were not altered within the whole investigation period (Table 3). RVEF remained at baseline level until onset of CPB in the HS-HES as well as in the HES group, whereas it decreased in the control patients (-17.3%). RVEDVI and RVESVI increased in both volume groups, but increased values were noticeable 40 minutes after volume application only in the HS-HES patients. At the end of the operation (45 minutes after termination of bypass) no more significant differences in haemodynamics occurred between any groups.

With regard to laboratory parameters, Hb and the number of platelets decreased in both volume groups (Table 4). None of the coagulation variables (fibrinogen, AT-III, TT, PT, PTT) differed significantly among the groups. Immediately after CPB, pulmonary gas exchange was significantly less compromised in patients who received HS-HES (+1.2%) than in those given HES (-14.5%) or the control patients (-19.7%) (Table 4). Blood viscosity was decreased only by HS-HES solution and remained almost unchanged in the other groups (Fig. 1). Serum sodium concentration increased only in the HS-HES patients, but never exceeded 155 mmol/litre (Fig. 2) and had already decreased below 147 mmol/litre on the first postoperative day. Immediately after volume application, osmolality was elevated significantly in the HS-HES patients compared to the control group, but no other group differences occurred in the further course of the investigation (Fig. 2). None of the patients suffered from renal failure, and free water clearance was similar in all groups (always below -80 ml/hour (Fig. 2)).

All patients had an uneventful course, and none suffered from sequelae attributable to the study.

Table 2. Changes in haemodynamic variables during the investigation. Data are presented as mean (SD).

Variable	Group	Baseline	After infusion	20 minutes after infusion	40 minutes after infusion	5 minutes after CPB	45 minutes after CPB
MAP; mmHg	HS-HES	78.5(7.8)	76.3(7.9)	78.2(9.5)	73.4(7.6)	81.7(9.1)	93.9(7.9)
	HES	80.0(7.7)	77.0(5.5)	81.4(8.8)	78.9(6.6)	80.2(9.1)	94.6(8.2)
	Control	77.3(7.9)	72.5(4.0)	78.9(8.0)	74.5(8.1)	81.1(9.3)	88.3(8.0)
HR; beats/minute	HS-HES	72.4(11.1)	74.4(8.8)	78.9(8.8)	78.9(7.7)*	90.6(9.0)	92.7(9.3)
	HES	72.4(7.7)	74.8(9.0)	80.7(9.0)	81.9(8.9)	91.0(7.9)	97.9(10.2)
	Control	71.9(9.1)	73.1(6.5)	79.6(9.1)	89.0(7.0)	99.0(8.4)	100.9(11.1)
PAP; mmHg	HS-HES	12.6(2.7)	15.9(3.0)	15.8(4.1)	15.8(2.7)	15.1(3.1)	16.3(3.0)
	HES	12.3(2.3)	16.2(2.8)	17.2(3.2)	14.6(3.1)	13.0(2.5)	15.9(1.9)
	Control	13.0(2.3)	13.8(2.2)	14.8(2.8)	12.3(2.9)	14.8(3.4)	14.7(2.6)
PCWP; mmHg	HS-HES	4.0(1.1)	8.2(1.5)*	7.8(1.9)*	7.4(1.5)*	6.4(2.0)	8.8(2.2)
	HES	4.2(1.4)	8.3(1.5)*	7.7(1.3)*	7.0(2.2)*	5.9(2.2)	9.1(2.9)
	Control	4.8(1.1)	3.1(0.7)	2.9(1.9)	2.1(2.2)	5.6(2.0)	8.1(3.0)
CI; (litres/minute)/sq m	HS-HES	3.0(0.4)	4.2(0.4)**	4.0(0.3)**	3.9(0.3)**	3.1(0.4)	3.1(0.6)
	HES	3.0(0.3)	3.8(0.4)*	3.5(0.3)*	3.7(0.3)*	3.2(0.6)	3.0(0.6)
	Control	3.2(0.3)	3.1(0.5)	2.7(0.4)	2.7(0.4)	3.0(0.5)	2.9(0.6)

MAP, mean arterial pressure; HR, heart rate; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index.

*p < 0.05 compared to control group; **p < 0.05 compared to HES and control groups.

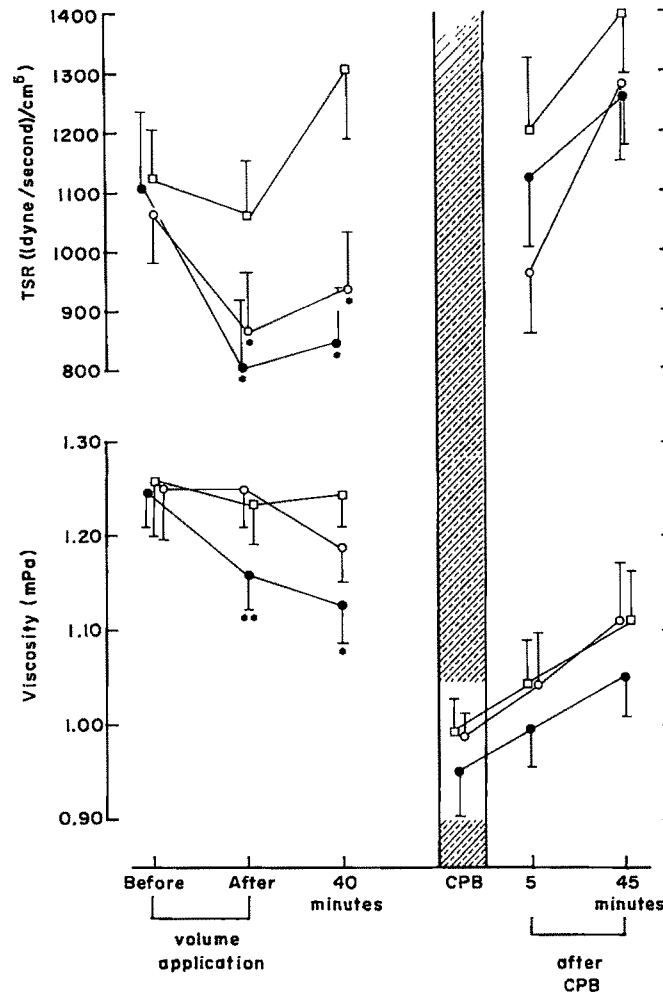


Fig. 1. Changes in total systemic resistance (TSR) and blood viscosity in the three groups.
* $p < 0.05$ compared to control group; ** $p < 0.05$ compared to HES and control groups.

Table 3. Changes in right ventricular haemodynamic variables. Data are presented as mean (SD).

Variable	Group	Baseline	After volume	20 minutes after volume	40 minutes after volume	5 minutes after CPB	45 minutes after CPB
RVPsyst; mmHg	HS-HES	21.9(3.3)	30.1(5.0)*	31.2(6.7)*	31.3(6.6)*	29.6(6.9)	29.4(5.4)
	HES	23.4(3.1)	28.0(4.9)*	32.0(8.1)*	30.4(8.8)*	33.7(8.9)	34.1(5.6)
	Control	23.7(3.6)	24.1(4.5)	26.2(6.3)	25.9(5.4)	28.3(6.6)	30.0(5.2)
RVEDP; mmHg	HS-HES	1.6(0.4)	2.0(0.7)	1.8(0.7)	2.4(0.9)	2.7(0.9)	3.3(1.3)
	HES	1.2(0.4)	1.6(0.5)	1.5(0.6)	1.8(1.2)	1.7(0.5)	3.1(1.3)
	Control	1.3(0.5)	1.3(0.4)	1.3(0.4)	1.7(0.8)	2.3(0.7)	2.9(1.2)
RAP; mmHg	HS-HES	1.7(0.8)	2.6(1.1)	2.5(1.3)	2.0(1.2)	3.9(2.1)	5.0(2.0)
	HES	1.5(0.5)	2.5(1.5)	2.3(1.2)	2.2(1.2)	2.9(2.1)	4.8(2.0)
	Control	2.0(1.0)	2.3(0.7)	2.5(1.1)	2.3(1.1)	3.3(1.4)	4.5(1.7)
RVEF; %	HS-HES	44.9(3.9)	45.8(6.6)	45.4(5.0)	45.3(5.0)*	43.1(8.8)	43.9(7.1)
	HES	47.6(4.0)	45.2(6.8)	47.5(7.7)	47.7(4.1)*	45.8(6.9)	45.0(10.0)
	Control	47.4(5.5)	45.8(7.5)	45.0(6.1)	39.4(4.6)	44.6(5.9)	39.9(5.0)
RVEDVI; ml/sq m	HS-HES	89.4(12.1)	120.9(22.8)*	110.1(26.1)**	105.3(19.1)**	85.1(18.1)	87.7(18.1)
	HES	93.2(11.9)	116.4(20.1)*	103.8(23.4)	83.1(18.9)	69.3(10.1)	68.5(13.2)
	Control	96.2(16.7)	94.8(12.9)	94.3(25.1)	93.3(16.7)	73.9(18.9)	63.8(19.4)
RVESVI; ml/sq m	HS-HES	50.2(17.4)	72.0(14.6)*	63.0(15.5)*	63.5(14.2)*	52.9(19.1)	54.3(13.4)
	HES	49.9(10.9)	67.3(18.2)*	56.3(13.2)	45.9(19.2)	36.6(11.1)	37.0(10.2)
	Control	53.8(15.3)	52.7(12.3)	51.8(23.1)	56.3(19.3)	40.6(13.4)	37.2(15.3)

RVPsyst, right ventricular systolic pressure; RVEDP, right ventricular end-diastolic pressure; RAP, right atrial pressure; RVEF, right ventricular ejection fraction; RVEDVI, right ventricular end-diastolic volume index; RVESVI, right ventricular end-systolic volume index.

* $p < 0.05$ compared to control group; ** $p < 0.05$ compared to HES and control groups.

Table 4. Changes in haemoglobin (Hb), number of platelets, pulmonary gas exchange, and red cell volume (RCV). Data are presented as mean (SD).

Variable	Group	Baseline	After volume	40 minutes after volume	5 minutes after CPB	45 minutes after CPB	5 hours after CPB	On the 1st postoperative day
Hb; g/dlitre	HS-HES	13.3(0.5)	10.8(0.6)*	11.0(0.6)*	9.1(0.7)	12.0(1.1)	13.2(1.1)	13.1(1.0)
	HES	13.3(0.8)	10.9(0.6)*	10.8(0.5)*	8.6(0.4)	12.0(1.0)	13.6(0.8)	12.0(1.0)
	Control	13.1(0.6)	13.0(0.7)	12.8(0.7)	9.8(0.8)	11.9(1.3)	12.0(1.2)	11.8(1.2)
Platelets; $\times 10^9$ /litre	HS-HES	184(31)	154(39)*	177(26)*	172(25)	139(18)	164(31)	152(33)
	HES	188(29)	158(22)	171(21)*	168(24)	139(29)	170(32)	150(40)
	Control	200(30)	197(24)	200(23)	166(33)	150(27)	144(28)	139(33)
PaO_2/FiO_2 ; kPa	HS-HES	47.7(5.6)	52.1(9.2)	51.6(6.7)	52.3(9.1)**	45.2(8.0)*	50.4(10.9)*	69.7(15.2)
	HES	46.8(8.8)	54.8(11.7)	54.7(9.7)	47.2(10.3)	41.3(7.3)	46.9(5.9)	65.3(16.3)
	Control	46.0(8.0)	49.9(8.8)	53.1(6.0)	43.2(11.7)	35.9(13.2)	38.4(8.9)	58.9(16.0)
RCV; (μm^3)	HS-HES	90.7(2.6)	91.5(2.6)	91.2(2.0)	91.6(2.2)	91.2(2.7)	91.7(2.7)	91.4(2.7)
	HES	90.3(3.0)	90.9(2.8)	91.4(3.0)	91.3(2.7)	91.0(3.3)	91.0(3.1)	91.1(3.1)
	Control	91.3(2.6)	91.6(3.0)	91.3(2.8)	91.3(3.5)	91.0(2.9)	91.3(3.0)	91.5(3.5)

* $p < 0.05$ compared to control group; ** $p < 0.05$ compared to HES and control groups.

Discussion

Fluid administration restores plasma volume and increases venous return to the heart, thus increasing cardiac output and improving haemodynamics.¹ The goal of the volume administration is to provide stable haemodynamics and to render both the macrocirculation and the microcirculation normal.¹⁶ Overload of the circulation and marked haemodilution should be avoided.

Cardiac surgery is a special situation in that a deterioration in microcirculation and an impairment in capillary integrity occur during the period of extracorporeal oxygenation.²²⁻²⁴ Large volumes of crystalloids, which are necessary to increase plasma volume, result in marked haemodilution with severe reduction in colloid osmotic pressure (COP) followed by impairment of pulmonary function in the post-bypass period.^{25,26} For these reasons, colloids are used more commonly for rapid restoration of

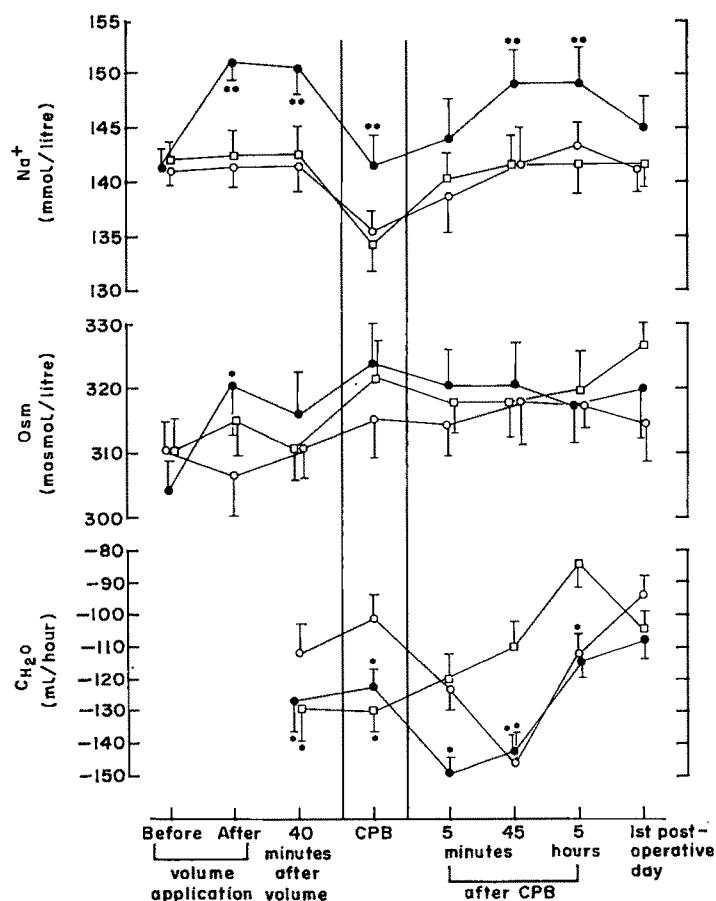


Fig. 2. Changes in serum sodium concentration (Na^+), serum osmolarity (Osm), and free water clearance (CH_2O) in the three groups. * $p < 0.05$ compared to control group; ** $p < 0.05$ compared to HES and control groups.

the circulating plasma volume, thus avoiding excessive fluid accumulation, particularly in the pulmonary tissue.

Recently, enthusiasm has been expressed for the use of hypertonic solutions in the treatment of refractory hypovolaemic shock.^{7-11,27} It was demonstrated that HS results in enhanced vasomotion and improved capillary flow,²⁹ effects that could not be achieved by conventional electrolyte or colloid solutions. The exact mechanism by which hypertonic solutions exert their beneficial haemodynamic effects are still controversial. Explanations include increased myocardial contractility, dilatation of the precapillary resistance vessels, and constriction of capacitance vessels.^{16,27,28} Animal studies have shown an increased rate of survival and superior cardiopulmonary function after resuscitation with these solutions in comparison to isotonic volume therapy.¹²

The use of extreme hypertonic solutions (up to 2400 mosmol/litre) has been the subject of little clinical investigation, and was studied mostly in severely burned patients.^{30,31} Thus, we were interested in comparing the effects of hyperosmolar volume application with HES 6% volume infusion in cardiac surgery patients.

One major finding from our study was that significantly less HS-HES solution than standard HES was effective in doubling PCWP before the start of cardiopulmonary bypass. Volume administration is often necessary in the pre-bypass period (as seen in the control group), because PCWP and CI decrease until the beginning of CPB. The haemodynamic effects of the HS-HES solution included an increase in CI, which was not transient, as has been reported when hypertonic saline solution was used alone;^{12,32} haemodynamics were stabilised for more than 40 minutes (until onset of CPB). In addition, the patients who received HS-HES solution required significantly less fluids, both during CPB and during the early postoperative period, indicating a long-lasting volume stabilising efficacy. Fluid balance during CPB seems to be of considerable importance with regard to organ function, and particularly pulmonary function.²⁶ Breckenridge *et al.*³³ noted that fluid accumulation occurs routinely during cardiopulmonary bypass and there may be a 33% increase in the measured extracellular fluid space postoperatively. Utley *et al.*³⁴ reported a fluid accumulation of 800 (ml/sq m)/hour of bypass. Bypass times in our study were moderate, and in longer cardiac surgical procedures a significantly more positive fluid balance is to be expected, with the risk of a deterioration in organ function. The hypertonicity of the HS-HES solution seems to allow utilisation of interstitial water to increase the intravascular volume,³⁵ thus reducing fluid input during CPB. On the basis of these findings, we believe that HS-HES solution might reduce tissue oedema formation. Intracellular fluid loss may also have beneficial microcirculatory effects,³⁶ particularly with regard to myocardial perfusion. Capillaries often show a swollen endothelium after a sustained period of ischaemia,³⁷ and this may result in reduced perfusion. Extraction of fluid from the endothelium in this situation will increase the inner diameter of the capillary, resulting in improved microcirculation.³⁶ Reduced viscosity in this situation, as seen in our patients, lowers the hydraulic resistance and might contribute to this improvement in myocardial blood supply.

Animal studies demonstrated an improvement in renal, intestinal, pancreatic, and myocardial flows after applica-

tion of 7.5% NaCl.^{16,38} We did not measure organ flows, but pulmonary gas exchange was significantly less compromised in the HS-HES than in the other groups, most likely because of the negative fluid balance during CPB in these patients. However, improvement in organ perfusion might have contributed to these effects. Renal function was not altered in HS-HES patients; free water clearance, which is said to be a reliable indicator of renal insufficiency after cardiac surgery, was lowest in this group.

Our results are difficult to compare with findings from others because of different study protocols; published reports cover a variety of hypertonic solutions over a wide range of volume and variable osmotic strength⁷⁻¹¹ (from 3% to 7.5% NaCl with osmolarity from 300 to 2400 mosmol/litre). Moreover, it is not clear how much species differences might affect the results.

In the present study, we used a hypertonic saline solution prepared in HES solution, whereas others applied HS with dextran to prolong haemodynamic effects.¹¹ Dextran, however, has well established anaphylactic and haemoreological effects.^{17,18} Coagulation is compromised during extracorporeal oxygenation procedures³⁹ and the effects associated with dextran are undesirable in cardiac surgery. HES, however, has minor effects on coagulation and is widely used in cardiac surgery.^{1,18} None of the patients treated with the new HS-HES solution showed impaired coagulation or anaphylactic reactions. However, the administration of such a hypertonic solution does pose certain theoretical risks; hypernatraemia and hyperosmolarity can result in cerebral dysfunction such as disorientation, confusion, and seizures by disruption of the blood-brain barrier.³⁸ No patient in our study suffered ill effects from the slightly elevated osmolarity (maximum 326 mosmol/litre). A serum osmolarity of 350 mosmol/litre has been reached in man without complications.⁴⁰ Moreover, elevated osmolarity might result in alterations in integrity of cellular elements of the blood, particularly of red cells. However, no negative effects were demonstrated with regard to red cells in our study; red cell volume (RCV) remained almost unchanged throughout the investigation period. Sodium concentration never exceeded 155 mmol/litre and was below 147 mmol/litre on the first postoperative day. Finally, application of hypertonic solution might result in circulatory fluid overload from an uncontrolled displacement of tissue fluid into the blood compartment. Correct assessment of fluid needs and determination of the appropriate volume therapy are often difficult. There is increasing evidence that the traditional methods for monitoring fluid therapy using central venous pressure and PCWP may be inadequate for regulating volume therapy and preventing fluid overload. A non linear relationship exists between volume and pressure particularly in cardiac patients. The compliance in these patients is often altered and monitoring of filling pressures alone is sometimes misleading in the evaluation of volume needs.¹

Patients who received HS-HES showed an increase in CI, an unchanged RVEF and an elevated RVEDVI, indicating a shift of Starling's curve to the right as the result of effective volume therapy. None of these patients had signs of fluid overload or showed a shift towards the descending limb of the ventricular function curve.

It is concluded from our study that hypertonic saline HES solution provides an interesting alternative volume therapy in cardiac surgery patients. Improvement in

haemodynamics was effective, superior to standard HES solution, and not transient. Fluid requirements were reduced significantly during and after cardiopulmonary bypass.

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Alkalinisation of prilocaine for intravenous regional anaesthesia

Suitability for clinical use

P. ARMSTRONG, J. WATTERS AND A. WHITFIELD

Summary

Eighty unpremedicated patients undergoing day-case hand surgery under intravenous regional anaesthesia were randomly allocated to receive, in a double-blind study, either 40 ml 0.75% prilocaine hydrochloride, with 5 ml 8.4% sodium bicarbonate or 5 ml 0.9% saline. The alkalinised group had significantly less pain on injection ($p = 0.0045$), during surgery ($p = 0.0074$) and 5 minutes after the tourniquet was deflated ($p = 0.0027$). The time elapsed between insertion of the block and commencement of surgery was not affected.

Key words

Anaesthetics, local; prilocaine.
Anaesthetic techniques, regional; intravenous.

Intravenous regional anaesthesia (IVRA) has been used since 1908¹ to produce anaesthesia of the hand and forearm. It has many advantages; it is reliable, simple to perform, has a controllable duration and a rapid recovery. This makes it ideal for day-case surgery. However, there are a number of disadvantages; onset of action can be slow resulting in inadequate anaesthesia or a prolonged wait before surgery; a tourniquet is needed which often creates considerable discomfort especially if inflation time is prolonged; the rapid recovery may result in pain soon after deflation, so that other forms of analgesia are required after operation; toxic reactions to the local anaesthetic may occur.

Various techniques have been used to prevent these complications. Tourniquet discomfort is reduced by fast surgery or the use of a double tourniquet.² The rapid recovery could be prevented by using bupivacaine,³ but because of its toxicity it is no longer approved for use in IVRA.⁴ Toxic reactions after tourniquet release may be reduced by repeated release and re-inflation of the cuff.⁵ An increase in the total local anaesthetic dose potentiates both the speed of onset of block and the anaesthesia produced, although the incidence of toxic side effects also increases. The maximum recommended dose of prilocaine is 4–5 mg/kg, although its concentration has varied from 0.5% to 2%.⁶ There is the possibility of methaemoglobin formation with the use of large doses of prilocaine.⁷

Alkalinisation of prilocaine by the addition of 8.4% sodium bicarbonate was shown to increase the speed of onset of anaesthesia and to prolong its duration after tourniquet release in IVRA in volunteers.⁸ This method therefore appears to have the potential to cause a block

without an increase in the amount of local anaesthetic used; this makes it both safer and more comfortable.

This study was designed to investigate the possibility that alkalinisation of prilocaine could result in an improvement in the clinical reliability of IVRA for hand surgery.

Methods

Eighty unpremedicated patients, aged 18 to 75 years, undergoing hand surgery as day cases were studied. Approval for the study was obtained from the local area ethics committee and full informed consent was obtained from all the subjects.

A standard technique was used in all patients. A 20-gauge cannula was inserted in a vein in the dorsum of the hand in the ulnar area, after placement of a cannula in the contralateral arm. A padded tourniquet was positioned around the upper arm, 5 cm proximal to the lateral epicondyle; the arm was exsanguinated with an Esmarch bandage and the tourniquet inflated to 300 mmHg. The bandage was then removed and the local anaesthetic solution injected over 45 seconds; the end of the injection was taken as time zero. Surgery started when the anaesthetist considered anaesthesia to be adequate. The tourniquet was released after completion of surgery, or after at least 20 minutes had elapsed since injection.

The local anaesthetic solution consisted of 40 ml 0.75% prilocaine hydrochloride to which had been added either 5 ml 0.9% saline (control group, pH = 6.40) or 5 ml 8.4% sodium bicarbonate solution (alkaline group, pH = 7.75). Allocation to each group was random (random numbers table) and the entire trial was carried out in a double-blind

Table 1. Patient data, mean (SD).

	Control group n=40	Alkaline group n=40
Age; years	48.6 (14.5)	48.7 (16.0)
Weight; kg	66.2 (8.7)	70.5 (15.4)
Males	18	15
Females	22	25

manner. It was previously determined that there was no precipitation of either mixture on standing for 48 hours.

The duration from time zero to the start of surgery (anaesthetic time), from the start of surgery to its completion (surgical time) and the total time the tourniquet was inflated (tourniquet time) was measured. The suitability of the hand for surgery was assessed by the anaesthetist using a 4-point scale (1 = poor, 2 = moderate, 3 = good, and 4 = excellent). The patient assessed the pain of injection of the local anaesthetic solution, the pain of surgery, the pain of the hand and arm just as the tourniquet was deflated, and the pain of the hand and arm 5 minutes after deflation, using a 10-cm visual analogue scale (VAS) with the left side labelled no pain and the right side the severest pain imaginable. The patient, in addition, assessed the state of the hand and arm before leaving the recovery room using a 4-point scale (1, full recovery, 2, moderate recovery, 3, slight recovery and 4, no recovery).

Student's unpaired *t*-test was used to analyse the patient data. The VAS, anaesthetic, surgical and tourniquet times and the 4-point scales were analysed by the Mann-Whitney test.

Results

Patient data are shown in Table 1. There were no differences between the groups. The surgical and tourniquet times were similar in both groups, which indicated similar operating conditions (Table 2). The anaesthetic time was similar; there was no decrease in the time required for anaesthesia to occur in either group.

Assessment of the state of the hand for surgery was similar, with both groups having good to excellent conditions (Table 3). Similarly, both groups' hands were in a similar state before returning to the ward; most had slight to moderate recovery. Blocks appeared sufficient for surgery in all but two cases (one in each group); some patients believed that they received poor anaesthesia but were happy to continue with the surgery.

Table 4 shows the pain scores. The pain of injection was statistically worse ($p = 0.0045$) in the control group as compared to the alkaline group. Similarly, the pain of surgery was greater ($p = 0.0074$) in the control group than the alkaline group. There was no difference in pain in the hand and arm at deflation between groups. The control

Table 2. The median (range) anaesthetic, surgical and tourniquet times (minutes).

Time	Control group	Alkaline group	
Anaesthetic	9.0 (4.0–19.0)	9.0 (5.0–15.0)	ns
Surgical	11.5 (3.0–30.0)	10.0 (2.0–26.0)	ns
Tourniquet	21.0 (20.0–43.0)	21.0 (20.0–40.0)	ns

Table 3. Median (range) scores assessed by a 4-point scale for the state of the hand, before surgery (1 = poor, 2 = moderate, 3 = good, and 4 = excellent), and before leaving the recovery room (1 = full recovery, 2 = moderate recovery, 3 = slight recovery and 4 = no recovery).

Arm state	Control group	Alkaline group	
Before surgery	3.30 (1–4)	3.52 (1–4)	ns
Leaving recovery room	2.55 (1–4)	2.35 (1–4)	ns

group had greater pain in the hand 5 minutes after surgery ($p = 0.0027$).

Two patients in the control group complained of a numb mouth and ringing in the ears on tourniquet deflation.

Discussion

The addition of 5 ml 8.4% sodium bicarbonate to 40 ml 0.75% prilocaine hydrochloride has an effect on the depth of anaesthesia provided. It decreases the pain of injection of the local anaesthetic mixture, produces a denser block with a decrease in the pain reported during surgery and prolongs the duration of the block as it reduces hand pain after tourniquet release. It has no effect on the state of the hand for surgery and causes no increase in the abnormality of the hand after surgery. It has, in addition, no effect on the time taken for the establishment of adequate anaesthesia after injection.

Alkalinisation of local anaesthetics has been attempted in both central and peripheral nerve blockade with partial success. Various local anaesthetics were used in epidural blockade. Lignocaine,^{9,10} 2-chloroprocaine^{11,12} and bupivacaine¹³ have all resulted in significantly improved blocks. Peripheral blocks that have been improved by alkalinisation include brachial plexus,¹⁴ sciatic nerve¹⁵ and peribulbar with bupivacaine¹⁶ and midtarsal blockade with mepivacaine.¹⁷ However, other studies were unable to demonstrate any advantages; these include alkalinisation of bupivacaine for epidurals,¹⁸ brachial plexus blockade¹⁹ and both mepivacaine²⁰ and bupivacaine²¹ for lower extremity blocks.

Why some trials have failed to demonstrate a positive result is unknown, although it may be because of the degree of alkalinisation of the local anaesthetic solution that is produced. Local anaesthetics have their site of action inside the nerve axon;²¹ if more drug is able to reach its site of action, then onset of action, density of block and duration

Table 4. Median (range) values for the visual analogue scores for pain in the hand and arm during injection of the local anaesthetic, surgery, just before tourniquet release, and 5 minutes after tourniquet release.

Pain score	Control group	Alkaline group	p value
Injection	0.70 (0–4.9)	0.15 (0–2.4)	0.0045
Surgery	0.60 (0–9.4)	0.05 (0–3.5)	0.0074
Before tourniquet release	0.05 (0–7.2)	0.00 (0–2.3)	ns
5 minutes after tourniquet release	0.30 (0–4.6)	0.00 (0–0.6)	0.0027

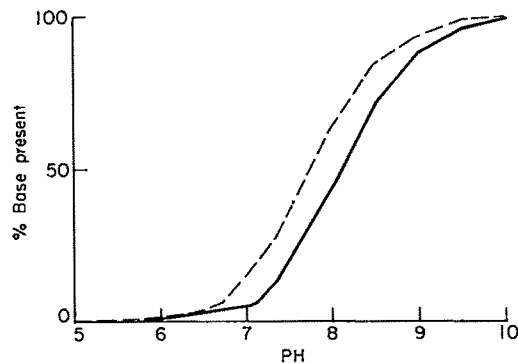


Fig. 1. Percentage amount of uncharged base present in solution at a given pH. Bupivacaine, —; prilocaine, - - - -.

of action will be greater, the 'mass effect'. Local anaesthetics are weak acidic salts and in solution exist in two forms, either charged (conjugate acid) or uncharged (base). The uncharged form is lipid soluble and is able to diffuse more easily across membranes than the charged form. The ratio of charged to uncharged drug is governed by the Henderson-Hasselbach equation.

$$(pK_a = pH - \log(\text{base}/\text{baseH}^+)).$$

The pK_a of the individual local anaesthetic drugs is fixed, but alkalinisation of the solution increases the amount of uncharged base present (Fig. 1). Unfortunately, raising the pH of the local anaesthetic solution too far results in precipitation of the drug. The pH at which this occurs varies with individual agents and whether adrenaline is added. Bupivacaine 0.5% precipitates above a pH of 7.0, although prilocaine is stable up to pH 8.0. Many of the above protocols only increased the pH of the solution slightly and therefore resulted in only a small increase in the amount of uncharged base present. It is probable that this increase is too small to have any effect on the 'mass effect'.

Raising the pH of 0.75% prilocaine hydrochloride (pK_a 7.75) from 6.40 to 7.75 increases the percentage of base present from 3 to 50%. This increase has resulted in a denser block, with less pain from surgery. In addition, it caused a prolongation of the anaesthesia of the hand. This is probably because of an increase in the amount of anaesthetic agent absorbed by the tissues as a result of their increased lipophilicity. Normally, after tourniquet release, 30% of the total dose is immediately released into the circulation, followed by a gradual release of the rest.²² It is likely if more is absorbed, less would be released as a bolus. In support of this, two patients experienced toxic signs on tourniquet deflation, both in the control group. However, in contrast to these positive findings, there was no increase in the speed of onset as measured clinically. However, this method of assessment is relatively crude and may have been insufficiently sensitive to determine true readiness for surgery accurately.

Interestingly, the pain of injection of the anaesthetic mixture was less in the alkalinised group. There are two possible factors to explain this. Firstly, the increase in pH may have reduced the pain. Secondly, there is an increase in the osmolarity of the alkaline solution that may have caused the effect.

In conclusion, alkalinisation of 0.75% prilocaine hydrochloride resulted in a clinical improvement in IVRA for hand surgery.

Acknowledgments

Dr P. Armstrong is a Research Fellow paid by the Association of Anaesthetists of Great Britain and Ireland.

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Flumazenil in ketamine and midazolam anaesthesia

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Summary

A double-blind, parallel group study using flumazenil and placebo was carried out to determine whether patients who received flumazenil would awake more quickly and whether this drug would reverse the protection conferred by midazolam on the psychic sequelae of ketamine. Fifty female patients were studied. The results showed that there was a significant reduction in awakening time ($p = 0.02$) and a very significant increase ($p = 0.001$) in the incidence of dreams in the flumazenil group.

Key words

*Antagonists, benzodiazepine; flumazenil.
Anaesthetics, intravenous; ketamine.*

Midazolam was shown to prevent the emergence phenomena and psychic sequelae associated with the administration of ketamine.^{1,2} We have previously described a method of total intravenous midazolam and ketamine anaesthesia for use in the field.³ This technique was found to be simple, effective and versatile and was adopted for general use in peace time, although a small percentage of patients have demonstrated delayed awakening. This is thought to be because about 5% of the population metabolise midazolam slowly.⁴ Patients would awaken more quickly if flumazenil was used to reverse the effects of midazolam, which is an essential feature of field anaesthesia. There is the fear, however, that the use of flumazenil may also reverse the protection conferred by midazolam on the psychic sequelae of ketamine.

The aim of this present study was to monitor the effects of flumazenil when given to patients who had received ketamine and midazolam for total intravenous anaesthesia with particular emphasis on the report of psychic symptoms including hallucinations.

Method

This was a double-blind, parallel group study to compare flumazenil and placebo after total intravenous anaesthesia with midazolam and ketamine. The patients were allocated to receive either flumazenil 0.2 mg or placebo according to a computer-generated randomisation code. Informed

consent was obtained in writing from all participating patients.

Fifty female patients aged between 16 and 60 years who presented for gynaecological surgery were recruited to the trial. The types of operation performed are listed in Table 1. Patients were ASA 1 or 2 and those with a history of psychiatric illness were not studied.

All patients received premedication of temazepam 20 mg 1 to 2 hours before operation. Anaesthesia was induced with midazolam 0.07 mg/kg, ketamine 2 mg/kg, alfentanil 0.015 mg/kg and vecuronium 0.1 mg/kg via a cannula in a vein on the dorsum of the hand. Maintenance was with an infusion of ketamine 200 mg, midazolam 5 mg and alfentanil 1.0 mg in 50 ml of normal saline at a rate of 0.5 (ml/kg)/hour until the completion of the operation. Patients' lungs were ventilated with air supplemented with oxygen

Table 1. Types of operation performed.

Abdominal hysterectomy	18
Vaginal hysterectomy	9
Abdominal sterilisation	10
Laparotomy	5
Vaginal repair	2
Salpingolysis	2
Laparoscopy	2
Cystodistension	1
Fenton's	1

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Accepted 20 April 1990.

Table 2. Demographic and infusion data. Values expressed as mean (SD).

	Placebo <i>n</i> = 25	Flumazenil <i>n</i> = 25
Age; years	35.9 (7.7)	34.8 (8.9)
Weight; kg	65.5 (13.9)	67.0 (13.8)
Midazolam; mg,		
induction	5.1 (2.0)	4.9 (1.5)
maintenance	5.0 (2.5)	4.6 (2.3)
Ketamine; mg,		
induction	131.0 (28)	134.0 (27)
maintenance	98.0 (46)	93.0 (47)
Alfentanil; µg,		
induction	982.0 (207)	1001.0 (208)
maintenance	948.0 (465)	928.0 (466)
Vecuronium; mg,		
induction	5.9 (1.4)	6.2 (1.4)
Time from induction to administration of test drug; minutes	56.2 (20.5)	54.6 (20.6)

throughout the procedure. Residual paralysis was reversed at the end with glycopyrronium 0.5 mg and neostigmine 2.5 mg followed by either flumazenil 0.2 mg or placebo.

The patients were taken to the recovery area after reversal where heart rate and arterial blood pressure were recorded after 5 and 15 minutes and at 15-minute intervals thereafter. The times to give accurately their name, date of birth, the place and the day were also recorded (as minutes after administration of the test drug). The patients on return to the ward were questioned as to whether they had had any dreams and, if so, whether they were pleasant or not; whether the anaesthetic regimen was to their satisfaction and whether they felt sick and (or) vomited at any time after their anaesthetic.

Statistical methods. The primary criterion of efficacy was the incidence of dreams on awakening from anaesthesia. The two treatment groups were compared using a Chi-squared test of independence for these data. Secondary efficacy criteria were the times to awareness, and the two groups were compared using the nonparametric Wilcoxon rank-sum test for these data.

Results

The two groups were comparable with respect to age, weight and ASA grade. Induction doses of midazolam, ketamine, alfentanil and vecuronium were broadly similar for the two groups, as were the maintenance doses. The times from induction to administration of test drug ranged from 20–90 minutes; the two treatment groups were again comparable in this respect (Table 2).

Intra-operatively there was a general trend for the heart rate, arterial blood pressure and end-tidal CO₂ to decrease

Table 4. Number of patients who experienced dreams, nausea, vomiting and acceptability of anaesthetic.

Variable	Placebo <i>n</i> = 25	Flumazenil <i>n</i> = 25
<i>Dreams</i>	2	13
pleasant	1	7
unpleasant	1	5
unspecified	0	1
Nausea	14	16
Vomited	5	5
Anaesthetic satisfactory	24	20

from the start of the operation; there was little difference between the two treatment groups. Oxygen saturation remained constant throughout the intra-operative period.

The arterial pressure increased slightly after operation compared with the final operative recording for the flumazenil patients, followed by a decrease over the initial part of the recovery period. The placebo patients in comparison showed a slight decrease in blood pressure from the last operative reading and little change over the recovery period.

Five flumazenil patients were reported as agitated, anxious or disorientated in the recovery room, with one of these possibly suffering hallucinations; only one placebo patient was reported to have shown signs of anxiety.

The majority of those patients who received flumazenil recovered slightly more rapidly; they gave their name, date of birth, the location and the day approximately 10 minutes quicker than those who received placebo. Only the difference in time to give the location accurately achieved statistical significance ($p = 0.02$).

Only two patients in the placebo group reported dreaming compared to 13 who received flumazenil ($p < 0.001$, Table 4). The dreams were pleasant in approximately half of these patients, including one who received the placebo.

There were few differences between the treatment groups with respect to nausea and vomiting. Overall 60% were nauseated, of whom one third actually vomited.

The majority of patients (88% overall) were satisfied with the anaesthetic regimen used. These were divided 96% placebo and 80% flumazenil, which was not significantly different statistically.

Discussion

The total intravenous method of anaesthesia using ketamine, midazolam and vecuronium has proved successful in the injured patient. The aim of anaesthesia in a disaster or field situation is to provide the minimum depth required for surgery and to obtund unwanted reflexes whilst preventing

Table 3. Times to awakening (minutes from administration of test drug).

Variable	Placebo mean (range)	Flumazenil mean (range)	Wilcoxon test	
			Z value	p value
Accurately report:				
Name	29 (5–72)	20 (3–56)	1.85	0.064
Date of birth	31 (8–72)	20 (5–59)	1.97	0.049
Place	34 (8–80)	22 (5–59)	2.33	0.020
Day	32 (9–74)	22 (7–64)	1.85	0.065

awareness and recall. Reversal and recovery must be rapid and complication free. Midazolam attenuates the emergence problems of ketamine but does prolong the action.

We omitted papaveretum and hyoscine for premedication and used temazepam in a previous study on female patients undergoing laparoscopy.⁵ We found this to be satisfactory and the patients preferred the nonpoid premedication. We supplemented the analgesic action of ketamine with alfentanil at induction and during maintenance. This combination of drugs was chosen for our present study.

Flumazenil was used successfully for the reversal of midazolam-supplemented general anaesthesia.⁶ Flumazenil reversed the effects of midazolam in this present study and some of the side effects of ketamine were unmasked. The incidence of dreams on awakening from anaesthesia was increased by a factor of 6.5 after flumazenil.

The maintenance infusion was continued until the end of the operation. This is a later end-point than that used in the two previous studies,^{3,5} but we wanted a repeatable end-point since we wished to ascertain if the group of patients who received flumazenil awakened more rapidly. The patients who were given flumazenil were fully awake, and could accurately give their name, date of birth, the day and their location somewhat earlier than placebo patients, but only the location provided significant difference between the two groups.

We used the patients' ability to give their name, date of birth, the day and their location as the most easily repeatable assessment of the patient being fully awake. The times to awakening were longer than might be expected and longer than we would accept in the field situation. However, in normal practice the maintenance infusion would be discontinued 10–15 minutes before the expected end of the operation.

Somewhat surprisingly, but fortunately, the anaesthetic technique used was found to be acceptable by 88% of the patients overall. There was no significant difference between the two groups in this respect.

We are no nearer the answer to the question whether midazolam has a specific action in the reduction of sequelae or whether this is the result of the amnesic and soporific effects of midazolam. In our previous report on ketamine, midazolam and isoflurane in patients who breathed spontaneously, there was one patient very slow to awaken,⁷ to whom we gave flumazenil; the patient awoke immediately. Flumazenil is an expensive drug and we would not recommend its routine use in patients who have received ketamine and midazolam anaesthesia. It should be given only to those who fail to awake promptly. It is, however, reassuring to know that even if there are apparent excitatory arousal phenomena these will not be dramatic and the patient is unlikely to complain.

Acknowledgments

We thank Dr P. Hooper of Roche Products Ltd for his expert advice and Dr S. Little for the statistical analysis of the results of this study.

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CASE REPORT

Long-term patient-controlled analgesia in children

M. J. MOWBRAY AND P. B. GAUKROGER

Summary

Three children who received patient-controlled analgesia for periods of up to 41 days are described. In each case patient-controlled analgesia allowed pain control to be achieved in difficult situations. No patient developed tolerance or clinical signs of dependence. This use of long-term patient-controlled analgesia warrants further evaluation.

Key words

*Analgesia; postoperative, patient-controlled.
Surgery; paediatric.*

Patient-controlled analgesia (PCA) is becoming well accepted in the management of acute pain in adults,¹ and was described in adolescent^{2,3} and pre-adolescent^{4,5} children. However, the reports in children describe only its short-term use, typically for the management of postoperative pain. Over 300 children aged as young as 5 years have received PCA in our hospital, with encouraging results. Three patients have received 'long-term' PCA.

Our method of prescribing PCA has already been described.⁵ We use the Graseby PCAS device with a 60-ml syringe attached to an intravenous line which incorporates a one-way valve. Morphine 1.0 mg/kg (maximum 60 mg) is made up to 60 ml with saline 0.9%. Initially, demand doses of 1.0 ml are given, with a 5-minute lockout interval. This regimen is often supplemented with a background infusion of 1.0 ml/hour. The volume of drug used, the quality of analgesia, and any sedation or nausea are assessed each hour by the ward nursing staff. A member of the acute pain service sees the patient at least once daily, and is also available 'on-call' for any after-hours problems.

Case histories

Case 1

A previously healthy 12-year-old girl (weight 40 kg) presented at another hospital with a large ischiorectal abscess. After incision and drainage, a large cavity remained that needed frequent packing and dressing changes. She was referred to our hospital 7 days after

surgery because inadequate analgesia had made these dressing changes impossible. Analgesia was initially with intravenous pethidine by bolus, but morphine syrup was used subsequently. Neither regimen produced acceptable pain relief; morphine also caused nausea and vomiting.

She was taken to the operating theatre after admission for examination under general anaesthesia. This demonstrated an abscess cavity that was slow to heal. PCA was started postoperatively using pethidine, since this had not caused nausea previously. Bolus doses of 10-mg were prescribed, with a 3-minute lockout interval and no background infusion. It was anticipated that this regimen would allow the patient to achieve adequate analgesia rapidly (because of the short lockout interval). Figure 1 shows the pethidine doses administered over successive 4-hour periods before and during 14 successive dressings, over a 9-day period. Severe pain was not reported on any occasion. Nausea was reported on only four out of a total of 216 hourly observations.

She presented for further curettage of the abscess cavity 5 months later. She requested PCA, which was provided, and analgesia was excellent for a period of one week while further dressings were applied.

Case 2

An 11-year-old boy who weighed 46 kg suffered 40% burns, mainly full-thickness, to his face, chest and hands. He received an infusion of morphine during his initial resuscitation, and presented for debridement and grafting of

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Accepted 12 March 1990.

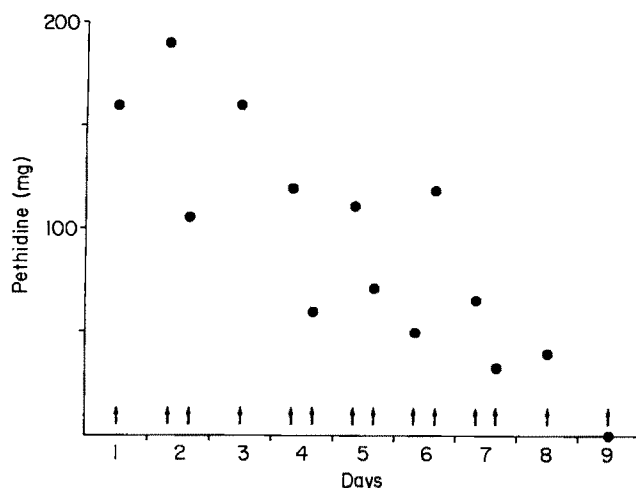


Fig. 1. Patient-controlled pethidine doses (●) for successive dressings. An arrow represents each dressing.

the burns 10 days later. PCA was started in the post-operative period and was used effectively for 20 days. Figure 2 shows daily morphine use, and demonstrates expected increases after surgery on two further occasions, followed by gradually declining requirements. The peak on day 9 represents a dose of 69 ($\mu\text{g}/\text{kg}$)/hour which is rather more than might have been prescribed by fixed dose. The patient did not complain of either moderate or severe pain at any time. Nausea never occurred.

Case 3

A 9-year-old girl (weight 30 kg) with cystic fibrosis presented with symptoms and signs of acute bowel obstruction. Laparotomy revealed a solid obstruction and a limited resection was performed. Postoperative analgesia was provided initially by an infusion of morphine, but this was ineffective despite increasing the infusion rate to 40 ($\mu\text{g}/\text{kg}$)/hour. The acute pain service was consulted on the first postoperative day and a routine morphine PCA system was started (Fig. 3). Analgesia improved rapidly and severe pain was not reported. This unfortunate girl subsequently underwent laparotomy four times over a 2-month period, and further bowel resections were required on each occa-

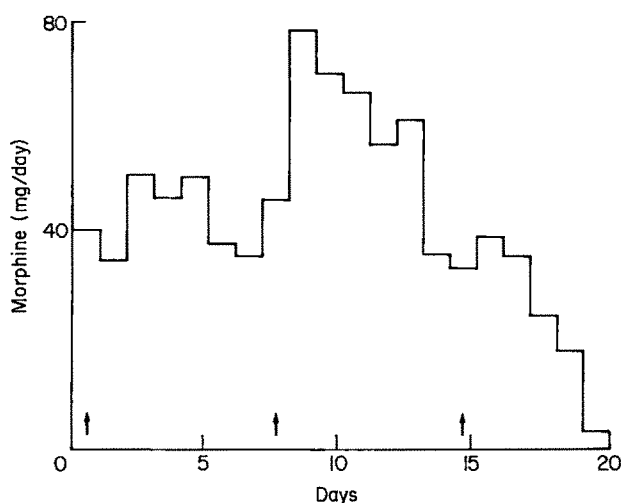


Fig. 2. Daily use of morphine. Surgery indicated by arrows.

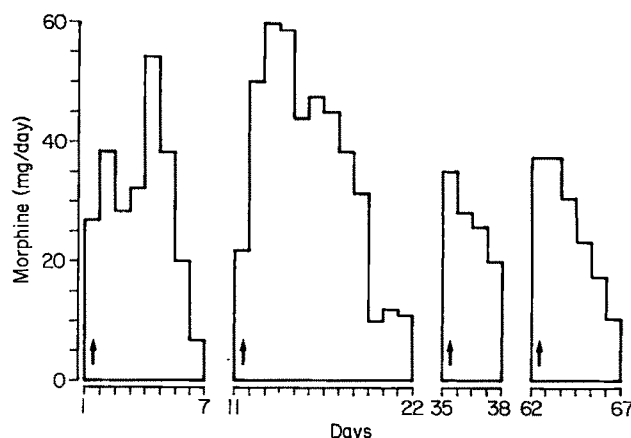


Fig. 3. Daily use of morphine. Surgery indicated by arrows.

sion. There was great difficulty in controlling colicky abdominal pain during the second period of PCA (days 11–22). We believe that this was because of continuing partial obstruction that eventually resolved with further surgery.

Discussion

PCA was used in these three patients to treat 'prolonged acute pain' rather than 'chronic pain'. Acute pain has a clearly identified organic cause that may not be present in chronic pain.

Previous reports²⁻⁵ of PCA in children have described its use for periods of up to 5 days after operation. We believe that this is the first report of 'long-term' PCA in children and that these three cases illustrate some of the features of the technique in this situation.

PCA has traditionally been prescribed to allow patients to titrate opioids against their perceived pain. Our first case illustrates how a patient can self-administer an opioid in anticipation of a painful procedure. This may seem to be difficult, but there is little reason to suppose that the patient's doctor could do it better. The absence of either pain or nausea suggests that fairly precise dosage was achieved. As might be expected, the second dressing in a day generally required less analgesia than the first. Furthermore, the dose required for the procedures decreased over several days. Both these observations imply that PCA was used appropriately by this patient.

Several interesting observations can be made from our second case. Firstly, wide fluctuations in analgesic requirements, associated mainly with surgery, can be accommodated easily with minimal side effects. Secondly, tolerance did not develop. Lastly, when the pain subsided, the patient stopped using PCA without prompting.

The patient in the third case benefited from the feeling of control that PCA gave her, but it was unfortunate that analgesia was not entirely satisfactory between days 11 and 20. Her pain during this time was colicky and related to persistent bowel obstruction. It has been our experience with other patients that colicky abdominal pain is controlled poorly by opioids.

Dependence can develop with long-term opioid use. However, there was no evidence clinically of dependence in any of our patients; all three reduced their own requirements as the pain subsided. Deliberate weaning was not necessary.

PCA will undoubtedly be employed more frequently in the postoperative period as it gains wider acceptance in children. It may be of benefit also in children whose pain management presents problems over longer periods. Further work is needed to determine precisely the value of PCA in the management of this type of patient.

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Anaesthesia for laparoscopic cholecystectomy using the Nd:Yag laser

The implications for a district general hospital

A. C. GREVILLE AND E. A. F. CLEMENTS

Summary

We describe the anaesthetic management of the first reported cholecystectomy by laparoscopic laser in the United Kingdom. The implications of the development of laser surgery in a district general hospital are discussed.

Key words

*Surgery; laparoscopy, cholecystectomy.
Equipment; laser.*

Lasers have been used in surgery for over 20 years. A laser is usually named after its active medium, the substance which emits the lasing action. The Nd:Yag laser is a solid crystal, Yttrium Aluminium Garnet, doped with Neodymium, a rare earth element. It is undergoing a rapid increase in use in gynaecological surgery and has now been used in general surgery to perform cholecystectomy via a laparoscope. The versatility of the Nd:Yag laser comes from its fiberoptic delivery, high power and contact probes for fine cutting and vaporizing. It has a number of potential advantages. Precise surgery can be performed using fewer instruments in the operative field; there is reduced blood loss and oedema, and sterilisation of the impact site; there is no interference with monitoring equipment; wounds are small, sufficient to carry the laparoscopic instruments; there is reduced postoperative pain and the potential for early discharge from hospital.¹

Case history

A 44-year-old, 72 kg woman was admitted to hospital for elective cholecystectomy. She had suffered two attacks of acute cholecystitis in the previous 7 months and subsequently chronic pain in the right hypochondrium. Ultrasonography demonstrated a 6-mm stone in the gall bladder. No other past medical history was noted. She was receiving no regular medication and was allergic to cotrimoxazole and penicillin. Examination revealed a healthy woman with mild epigastric tenderness to deep palpation. Full blood count and urea and electrolytes were within normal limits.

Premedication consisted of pethidine 75 mg and promethazine 12.5 mg intramuscularly one hour before anaesthesia. Anaesthesia was induced with thiopentone 275 mg and fentanyl 100 µg, after intravenous atropine 0.3 mg,

and the trachea was intubated with an 8.0 mm Portex oral tube after suxamethonium 100 mg had taken effect. An infusion of compound sodium lactate solution was started and a urinary catheter inserted. The patient's eyes were protected with moistened gauze underneath aluminium foil. A nasogastric tube was passed and the patient was placed in a head-up position, in order to provide optimum conditions for laparoscopic surgery.

Anaesthesia was maintained with enflurane and nitrous oxide in 40% oxygen and muscular relaxation with atracurium 35 mg followed by an infusion at 0.5 (mg/kg)/hour. Fentanyl was given in 50-µg boluses to provide analgesia, up to a total of 450 µg.

Monitoring consisted of electrocardiography, pulse oximetry, end-tidal capnography, inspired oxygen fraction, ventilator inflation pressure, nasopharyngeal temperature, and urine output; automatic blood pressure measurements were made at 3-minute intervals.

The operative procedure lasted 3.5 hours. An 11-mm subumbilical skin incision was made to accommodate the laparoscope, and four 5-mm abdominal incisions were made for the instruments. Between 900 and 1350 litres of carbon dioxide (two full cylinders and one part full, unweighed cylinder) were used to sustain the pneumoperitoneum. End-tidal capnography was in the range 4-5.6 kPa, with stable ventilation variables. An operative cholangiogram was performed. The cystic duct and cystic artery were clipped with titanium clips. The surgeon encountered two bleeding vessels during the gall bladder dissection from the liver using the Nd:Yag laser, and haemostasis was achieved using diathermy. Blood loss was difficult to assess from the laparoscopic view relayed with 6 times magnification onto a television screen, but pulse and arterial blood pressure measurements were virtually unchanged.

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Accepted 26 April 1990.

Polygeline 1000 ml and 3700 ml of crystalloid were given as intra-operative fluid replacement.

The gall bladder was removed complete through the largest incision as the laparoscope was withdrawn. Reversal of neuromuscular blockade was achieved after skin closure, using neostigmine 5 mg and glycopyrronium 1 mg. Recovery from anaesthesia was uneventful and rectal diclofenac 100 mg was administered before the patient's discharge from the recovery ward.

Her postoperative course was complicated by haematoma formation. She was discharged on the 5th post-operative day, but readmitted one week later for laparotomy to evacuate the haematoma.

Discussion

The anaesthetic management of this case was complicated by the combination of prolonged laparoscopic surgery and the Nd:Yag laser. The use of a camera has advantages for the anaesthetist because the operative field is in view. The disadvantage is that because of the magnification it is difficult to assess blood loss.

The vast amount of carbon dioxide used was alarming. Some of it must have entered the operating theatre atmosphere through the five abdominal incisions and from the connexions on the laparoscope itself; some would also be removed by suction. For upper abdominal laparoscopic surgery the pneumoperitoneum required is substantially greater than that required for pelvic diagnostic laparoscopy, when 2–3 litres of carbon dioxide are usually sufficient. An additional complication was the presence of 17 people in the operating theatre: there were laser operators, representatives of the equipment manufacturers and a television crew, as well as the surgical and anaesthetic teams.

This case report provides a useful forum for discussion of the impact of the Nd:Yag laser on a district general hospital and the implications for an anaesthetic department. The anaesthetist is isolated within the operating theatre, the doors of which must remain locked while the laser is in use. All windows must be laser-proofed with dark, nonreflective materials, which compounds the isolation. The anaesthetist may be unable to attend the recovery room or other operating theatres in a suite for relatively

long periods, and allowance for this must be made in the deployment of other anaesthetic staff.

The importance of anaesthetic monitoring equipment must be emphasised. The operating theatre is darkened, surgery prolonged and all personnel are required to wear protective goggles which may be tinted. Observation of the patient's colour can be misleading under these conditions. Capnography is vital because of the amount of carbon dioxide used.

The financial implications to a health authority of developing this type of service are multifarious. There is the capital cost of the Nd:Yag laser, camera and the associated pelviscopic and hysteroscopic equipment (£144 000); the cost of laser maintenance and the supply of new fibres; the need for appropriately trained staff to operate the laser; increased patient turnover due to early discharge from hospital, and the additional cost of treating patients referred from other health authorities. Moreover, the 'laser proofing' of an operating theatre used for routine gynaecological and general surgery may involve significant changes to pre-existing layouts. New doors may be required to isolate the theatre and alarms, locks and intercoms fitted.

The impact of the Nd:Yag laser on a district general hospital is potentially enormous as its use becomes more common in various surgical specialties. It is becoming more frequently used in this hospital in gynaecology for endometrial ablation via a hysteroscope, and laparoscopic surgery; many patients are treated as day cases. It has also been used in urology via a flexible cystoscope. There is considerable disruption to the operating theatre timetable because at present only one theatre is 'laser proofed'. The ideal for the future would be that all theatres are suitably equipped for Nd:Yag laser use.

Acknowledgments

We thank Mr P. Frecker and Mr D.L. McMillan for permission to publish this report.

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CASE REPORT

Life-threatening complications during anaesthesia in a patient with a ventriculo-atrial shunt and pulmonary hypertension

P. J. BUTLER, R. A. WHEELER AND P. M. SPARGO

Summary

A 6-year-old patient with hydrocephalus who underwent revision of a ventriculo-atrial shunt is described. Anaesthesia was complicated by the occurrence of systemic hypertension and arterial hypoxaemia. The patient was subsequently found to have pulmonary hypertension secondary to recurrent pulmonary thromboembolism. The pathophysiological mechanisms for the patient's deterioration are discussed and the anaesthetic management of children with pulmonary hypertension is outlined. It is concluded that patients with a ventriculo-atrial shunt who present for surgery should be screened carefully for the presence of pulmonary hypertension.

Key words

Anaesthesia; neurosurgical, cerebrospinal fluid shunt.

Lung; pulmonary hypertension.

The most commonly employed surgical treatment of hydrocephalus involves insertion of a shunt from the cerebral ventricle to the right atrium or peritoneal cavity. The peritoneal route has become marginally more popular since the development of the slit-valve catheter, which incorporates a valve at the distal end which opens at a predetermined pressure. Ventriculo-peritoneal shunts are prone to blockage while ventriculo-atrial shunts have an increased risk of both infection and pulmonary hypertension (PH). The effects of PH on right and eventually left ventricular function lead to considerable morbidity and mortality¹ and present major anaesthetic difficulties. We describe a patient who underwent revision of a ventriculo-atrial shunt and who developed life-threatening complications under general anaesthesia.

Case history

A 6-year-old girl was admitted for revision of the proximal end of her ventriculo-atrial shunt. She had suffered from intermittent vomiting and headaches during the preceding month. Surgical repair of a meningomyelocele, diagnosed at birth, was not performed because of absent leg function, but a ventriculo-atrial shunt was inserted at 6 months of age for progressive hydrocephalus. She also suffered from

recurrent urinary tract infections, and an ultrasound scan revealed a left hydronephrosis and hydroureter. A left nephrectomy was to be carried out at a later date.

The patient weighed 16 kg, had gross hydrocephalus and was paraplegic, although otherwise normal neurologically. She was alert and talkative. Routine pre-operative assessment revealed a heart rate of 140 beats/minute and a systolic murmur heard throughout the precordium; these had not been noted at any of her many previous admissions. The initial clinical and echocardiographic findings were thought to be consistent with a ventricular septal defect and antibiotic cover was advised by a paediatric cardiologist. An intravenous cannula was inserted to the dorsum of the hand after premedication with EMLA cream, and thiopentone 5 mg/kg was administered. Tracheal intubation was performed after administration of vecuronium for neuromuscular blockade. The oxygen saturation was noted to be 60% immediately after intubation despite administration of 100% oxygen. Correct position of the tracheal tube was confirmed initially by auscultation and then by laryngoscopy and re-intubation. The heart rate was 100 beats/minute and arterial pressure measured noninvasively in the right arm was 70/40 mmHg. Arterial pressure then became unrecordable and atropine 0.3 mg was given intravenously followed by ephedrine 1.5

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Accepted 2 April 1990.

mg. Systolic pressure improved to 90–100 mmHg and heart rate to 130–160 beats/minute. Anaesthesia was maintained with 1–2% enflurane in oxygen and arterial saturation was between 82 and 90% for the remainder of the operation. Arterial oxygen saturation improved to 95% after resumption of spontaneous breathing, and the tracheal tube was removed without incident.

Doppler echocardiography with colour flow mapping, performed later, revealed a dilated right heart but no evidence of an intracardiac shunt. Moderate to severe tricuspid regurgitation was noted with right ventricular systolic pressure close to systemic values. There was no right ventricular outflow tract obstruction. A ventilation-perfusion scan demonstrated multiple filling defects in both lung fields consistent with pulmonary emboli.

It was decided to remove the ventriculo-atrial shunt in order to eliminate the source of further pulmonary embolism and thereby halt the progression of pulmonary vascular disease. The creation of a ventriculo-peritoneal shunt was also necessary to control intracranial pressure. Electrocardiograph electrodes and a pulse oximeter probe were applied in the anaesthetic room before induction, which was achieved with fentanyl 0.1 mg/kg and vecuronium 2 mg via an intravenous cannula. The lungs were ventilated by mask with 50% oxygen in nitrous oxide before tracheal intubation. The heart rate was 90 beats/minute and oxygen saturation remained at 95%. However, the patient developed a bradycardia of 45 beats/minute immediately after tracheal intubation, and oxygen saturation decreased to 77%. Second degree atrioventricular block with Wenckebach phenomenon (Mobitz type 1 block) then developed. Intravenous atropine 0.2 mg restored sinus rhythm at a rate of 150 beats/minute and oxygen saturation returned to 90–95% over the next 5 minutes. Removal of the ventriculo-atrial shunt and insertion of a ventriculo-peritoneal shunt proceeded uneventfully. Spontaneous breathing was restored after reversal of neuromuscular blockade with neostigmine and glycopyrronium, and oxygen saturation remained around 90% on room air after removal of the tracheal tube.

The patient has since developed cor pulmonale with hepatomegaly, ascites and ankle oedema. No further surgery is planned.

Discussion

Ventriculo-atrial shunts are performed less frequently nowadays because of the risk of thrombus formation and multiple microemboli from the catheter tip. Pulmonary hypertension due to recurrent embolism with or without cor pulmonale is well described.^{2–4} However, there is little, if any, information concerning the anaesthetic hazard which patients with a ventriculo-atrial shunt present. Brief mention is made in a recently published textbook of the risk of developing pulmonary hypertension, but the hazard of general anaesthesia in this group of patients is not discussed.⁵ Histological evidence of pulmonary embolism can be found in over 50% of patients with a ventriculo-atrial shunt, although histological evidence of pulmonary hypertension is uncommon.⁶

The sudden deterioration in arterial pressure and arterial oxygen saturation after induction of anaesthesia in our patient is attributable to several causes. The total body weight of hydrocephalic patients is a poor guide to the drug

dose required because the head makes a disproportionately large contribution to overall body weight. This led, in our patient, to a relative overdose of thiopentone during the first anaesthetic. Barbiturates cause myocardial depression and a reduction in venous tone, both of which result in decreased cardiac output.⁷ The direct effect of barbiturates on pulmonary vascular resistance (PVR) is probably not significant.⁸ However, a number of other factors affect PVR adversely. Induction of anaesthesia results in decreased functional residual capacity which in turn is associated with a higher PVR than in the unanaesthetised state. Nitrous oxide increases PVR in children with increased pulmonary blood flow.^{9,10} Controlled ventilation also increases PVR by raising mean intrathoracic pressure. Hypoxaemia and acidosis also increase PVR.¹¹ An elevated PVR leads to an increase in right ventricular afterload. The ejection fraction decreases because changes in right ventricular contractility are limited by the thin right ventricular free wall.¹² Right ventricular output may be improved initially by increasing preload (Frank-Starling law) but ultimately this has adverse consequences. Right ventricular end-diastolic volume and right ventricular end-diastolic pressure (RVEDP) increase and according to Laplace's law, myocardial wall tension also increases. Coronary blood flow is decreased because of the limitation of right ventricular coronary blood flow (which under normal circumstances occurs in systole *and* diastole) to diastole only. The increased wall tension and decreased coronary flow result in subendocardial ischaemia. Volume overload of the right ventricle exacerbates tricuspid regurgitation and results in a reduction in forward flow. Finally, increases in RVEDP also cause a shift in the interventricular septum which impairs left ventricular function.

Thus, the many interrelated factors that contrive to impair right and left ventricular function and lead to decreased cardiac output are responsible for the high morbidity and mortality associated with pulmonary hypertension.¹ Arterial desaturation was less marked in our patient during the second anaesthetic, possibly because thiopentone was avoided and an opioid was used. Gentle assisted ventilation was thought essential to maintain oxygenation but was also desirable because of the possibility of raised intracranial pressure. It might have been prudent to avoid nitrous oxide altogether and to use a volatile agent. Enflurane may cause a slight elevation in PVR but halothane and isoflurane appear to have little effect.⁸

The pathophysiology outlined above suggests that it may be unwise to give a volume load to improve venous return. Further right ventricular distension only increases subendocardial ischaemia, increases interventricular septal shift and worsens myocardial function.¹³ The main goal of therapy is to increase aortic pressure and thus right ventricular myocardial blood flow.¹³ This reverses myocardial ischaemia and improves right ventricular function even in the face of continuing elevation of right ventricular afterload. It is preferable to choose an inotropic agent that does not increase PVR.¹ Clearly there is no ideal agent. Noradrenaline is probably the most suitable drug in these circumstances because it increases systemic vascular resistance without causing a tachycardia. It was shown to decrease PVR in animal studies.¹⁴ We chose ephedrine because it was available immediately. It has some vasoconstrictive effect, but acts mainly by direct cardiac stimulation to improve contractility and thus cardiac output.¹⁵

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CASE REPORT

Nitrobenzene poisoning and spurious pulse oximetry

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Summary

The successful management of nitrobenzene poisoning in a 21-year-old patient is presented. We report our experience of ventilatory care with additional intravenous methylene blue and ascorbic acid therapy. Pulse oximeters available at present are not useful in patients treated with methylene blue and should be used cautiously in the presence of cyanosis of unknown aetiology.

Key words

Equipment; pulse oximeter.

Complications; methaemoglobinaemia.

Several dyes are used in screen painting. They may be ingested either accidentally or in attempted suicide and cause acute toxicity by inducing methaemoglobinaemia and dilatation of smooth muscle (especially vascular smooth muscle).¹ The conversion of haemoglobin (HbFe^{2+}) to methaemoglobin (HbFe^{3+}) reduces tissue oxygenation since methaemoglobin cannot carry oxygen and the presence of oxidised iron changes the haem tetramer in such a way as to reduce oxygen release in the tissues. The arterial oxygen saturation, as measured by pulse oximeter, may be erroneous because of the presence of methaemoglobinaemia itself as well as the dye used in the treatment. A case of poisoning with nitrobenzene (a dye used in screen painting) is described in this report.

Case history

A 21-year-old man engaged in a screen painting business was brought to the intensive care unit in a deeply comatose state with very shallow breathing. An alleged history of taking 30–40 ml of 'varnish' (a nitrobenzene dye used in screen painting) 30 minutes before admission was obtained from the relatives. He had both peripheral and central cyanosis. Examination revealed both pupils to be of normal size and reacting to light, a regular heart rate of 160 beats/minute, blood pressure of 80/54 mmHg and respiratory rate of 28/minute. No other anomalies were detected in the cardiovascular system. His chest was clear on auscultation.

Immediate tracheal intubation was carried out and inter-

mittent positive pressure ventilation (IPPV) instituted with Fio_2 1.0, tidal volume 500 ml and ventilatory rate 14/minute. An intravenous line was established and blood samples drawn for investigations were dark brown in colour. Arterial blood gases during IPPV with 100% oxygen showed pH 7.42, base excess -4.3 mmol/litre, Pco_2 4.4 kPa and Po_2 51.2 kPa. Continuous electrocardiograph (ECG) and noninvasive blood pressure monitoring were undertaken. Oxygen saturation (Spo_2) measured using an Ohmeda Biox 3700 pulse oximeter indicated that the Spo_2 was 70–74%. Central venous pressure (CVP) was 7 cmH_2O . A drop of blood put on a white filter paper revealed a chocolate brown colour, and when oxygen was bubbled through the sample there was no change in the colour. A diagnosis of methaemoglobinaemia secondary to nitrobenzene ingestion was made.

Gastric lavage was performed with 5 litres of normal saline and hypotension was treated with positioning and intravenous fluids. The patient became conscious, well orientated and started responding to verbal commands after one hour of mechanical ventilation. His heart rate decreased to 120/minute, blood pressure increased to 100/70 mmHg and CVP was maintained at 9 cmH_2O . Cyanosis was now evident peripherally although central cyanosis was also obvious. Fio_2 was reduced to 0.7; Spo_2 fluctuated between 74 and 81%. The serum methaemoglobin concentration was 4.29 g/dlitre (37.2% of total Hb).

A slow intravenous infusion of ascorbic acid 1 g in 5% glucose twice a day was started. A solution of methylene

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Accepted 12 March 1990.

blue 1% was prepared and 60 mg (1 mg/kg body weight) was injected intravenously over a period of 5 minutes. The colour of the patient changed dramatically from brownish blue to pink 35 minutes after this injection. Initially SpO_2 decreased from 74% to 68–70% and 35 minutes later it increased to 84–85% (FiO_2 0.7). The pulse rate decreased to 99/minute and blood pressure increased to 106/74 mmHg. The urine was found to be bluish in colour. A further dose of methylene blue (50 mg) was administered after 50 minutes; soon after this infusion, SpO_2 decreased from 84% to 80%. IPPV was continued, and 25 minutes later SpO_2 improved to 88% (FiO_2 0.7). Two units of packed red cells were crossmatched and transfused.

A peripheral blood smear showed evidence of haemolytic anaemia, but urine examination revealed no occult blood. Methaemoglobin measurement was repeated 2 hours after the second dose of methylene blue, and was 0.6 g/dlitre (5.7% of total Hb).

FiO_2 was reduced to 0.5 12 hours after admission; SpO_2 was maintained at 86–89%. Assisted spontaneous ventilation was used successfully with FiO_2 0.4 on the next day, and SpO_2 was in the range 89–91%. The trachea was extubated 38 hours after admission. SpO_2 was maintained at 91–93% (FiO_2 0.4). Arterial blood gas studies revealed pH 7.41, PCO_2 5.3 kPa, PO_2 23.8 kPa and base excess +1 mmol/litre. Arterial oxygen saturation was 99.2% (c.f. SpO_2 91–93%) as calculated from the PO_2 . Oxygen administration was discontinued 6 hours after extubation and the patient was discharged on the fifth day of admission.

Discussion

Methaemoglobin is produced in small quantities by oxidation of the ferrous porphyrin complex, but under normal circumstances this ferric form is promptly reduced, thereby keeping the level of methaemoglobin in the blood at a low level.² Nitrates and some other substances interfere with this process and allow methaemoglobin to accumulate; in addition, nitrates oxidise haemoglobin to methaemoglobin in cases of severe poisoning. They are metabolised in the liver by nitrate reductase (glutathione dependent) to glyceryl dinitrate and nitrite. The nitrite is thought^{3,4} to convert haemoglobin (HbFe^{2+}) to methaemoglobin (HbFe^{3+}). Methaemoglobin reduces tissue oxygenation by two mechanisms:¹ iron in the ferric rather than ferrous form is unable to combine with oxygen and consequently the oxygen-carrying capacity of the blood is reduced; the presence of oxidised iron changes the haem tetramer in such a way as to reduce oxygen release in the tissues (i.e. shifts the oxyhaemoglobin dissociation curve to the left as in alkalosis).

Methaemoglobin concentrations between 30% and 50% of total haemoglobin produce moderate depression of the cardiovascular and central nervous systems.¹ This was manifest in our patient as stupor, tachycardia, hypotension and respiratory depression. Methaemoglobin is a dark pigment that causes venous blood to appear chocolate brown. Bubbling oxygen through it does not change the colour. A crystal of potassium cyanide added to a dark venous sample diluted 1/100 with deionised water turns the sample pink because the cyanohaemoglobin complex thus formed is pink in colour.

In methaemoglobinaemia, a gap exists⁵ between the

calculated oxygen saturation (from PO_2) and oxygen saturation measured by pulse oximetry. PO_2 is normal in this condition.

Pulse oximetry readings in methaemoglobinaemia are spurious both because of methaemoglobin and because of methylene blue, which is used as part of the treatment.^{5–11} In such conditions, pulse oximetry readings can be unexpectedly and misleadingly low (68–74% on admission in the present case). The presence of 37.2% methaemoglobin which has an absorbance similar to that of reduced haemoglobin at a wavelength of 660 nm, causes an increase in the absorbance ratio at 660/940 nm.¹¹ This is interpreted as a decrease in % HbO_2 and results in an SpO_2 reading below 100% (68–74% in this case). Methylene blue is known to cause erroneous SpO_2 measurements because this dye has an absorbance peak at a wavelength of 668 nm.^{6–8} In the present case, a transient decrease in SpO_2 was noticed on both occasions that methylene blue was infused (from 74% to 68% and from 84% to 80%), but 35 minutes after the first dose of methylene blue, SpO_2 started to increase due to dilution of the dye and its rapid renal clearance. These drawbacks of pulse oximetry are well known.^{5–11}

Under these conditions, pulse oximetry underestimates arterial or functional saturation, but overestimates fractional saturation or oxyhaemoglobin fraction according to the definition of Zijlstra and Oeseburg.^{12–14} Such patients should be monitored using arterial blood gases and there is little point in persisting with pulse oximetry or reliance upon it as a guide to the inspired oxygen concentration.

In the presence of methaemoglobinaemia, initial attention should always be directed towards improvement in oxygen delivery. In this patient, conversion of methaemoglobin to haemoglobin was hastened by administration of the reducing agents ascorbic acid and methylene blue. Ascorbic acid infusion results in acidosis, and a resultant shift of the oxygen dissociation curve to the right which improves oxygen delivery to the tissues. Methylene blue (tetramethylthionine chloride) is the antidote of choice for methaemoglobinaemia.¹ The recommended dose is 1 mg/kg of methylene blue 1% intravenously over a period of 5 minutes.¹⁵ At high levels of methaemoglobin, methylene blue reduces the half-life of methaemoglobin from 15–20 hours to 40–90 minutes.¹⁶ Methylene blue acts as a cofactor to increase the erythrocyte reduction of methaemoglobin to oxyhaemoglobin in the presence of NADPH, utilising the hexose monophosphate shunt pathway. The methylene blue is oxidised to leukomethylene blue, which is the electron donor molecule for the nonenzymatic reduction of methaemoglobin to oxyhaemoglobin.¹⁷

Acknowledgments

The authors thank Professor N. Madan, Department of Clinical Pathology for her invaluable guidance in the measurement of serum methaemoglobin levels. Thanks also go to Ms H. Kaur for her secretarial assistance.

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CASE REPORT

Dural puncture during attempted stellate ganglion block

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Summary

Dural puncture is rarely reported as a complication of stellate ganglion blockade. Breach of the dura occurred in the case described because of the anatomy of the cervical spine.

Key words

Anatomy; stellate ganglion, subarachnoid space.

Dural puncture is a rare complication of stellate ganglion block when it is performed by the anterior (paratracheal) approach and detailed accounts of subarachnoid injection (by any route) are infrequent.¹⁻⁴ Many large series of stellate ganglion blocks using the anterior route have been reported without this complication.^{3,5-7} The following case is of interest since it demonstrates how the dura may be breached, even with apparently careful technique.

resulted in free backflow of 2-3 ml of clear fluid presumed to be CSF. The procedure was abandoned. Drug therapy with amitriptyline and sodium valproate was started.

Case history

A 40-year-old female presented with a 4-year history of pain in the neck and left arm radiating to the hand. She described a painful burning sensation associated with heaviness and paraesthesia. She also described intermittent swelling of the hand. The pain had not responded to oral analgesics or to traction of the cervical spine. She was otherwise fit and healthy with no significant past medical history. She was investigated fully by the neurosurgical team before referral. A cervical myelogram was normal (Fig. 1). The patient, on examination, was found to be moderately obese. No neurological deficit was detected in her neck or arm, and her history seemed typical of a sympathetically mediated pain. The treatment options were discussed with her and it was decided to perform a stellate ganglion block. The patient was positioned in the standard manner for the anterior approach to the ganglion. The transverse process of C₆ was palpated and the needle introduced perpendicular to the skin and advanced until bone was encountered. Routine aspiration before injection

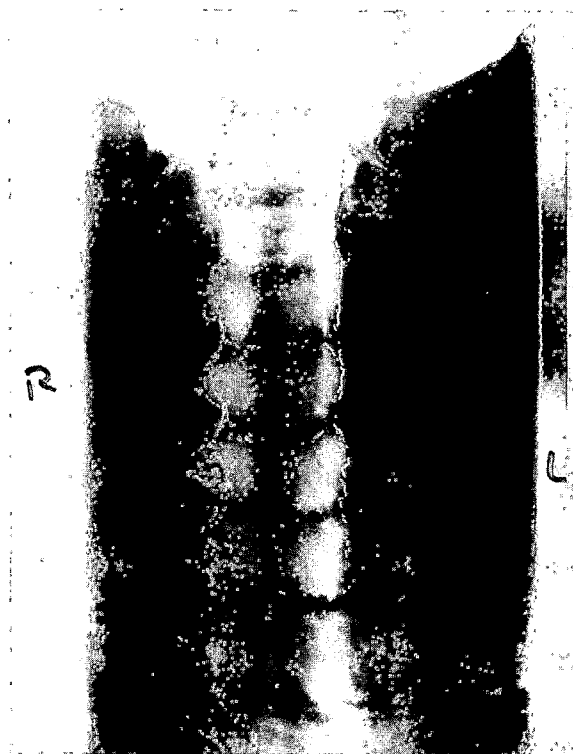
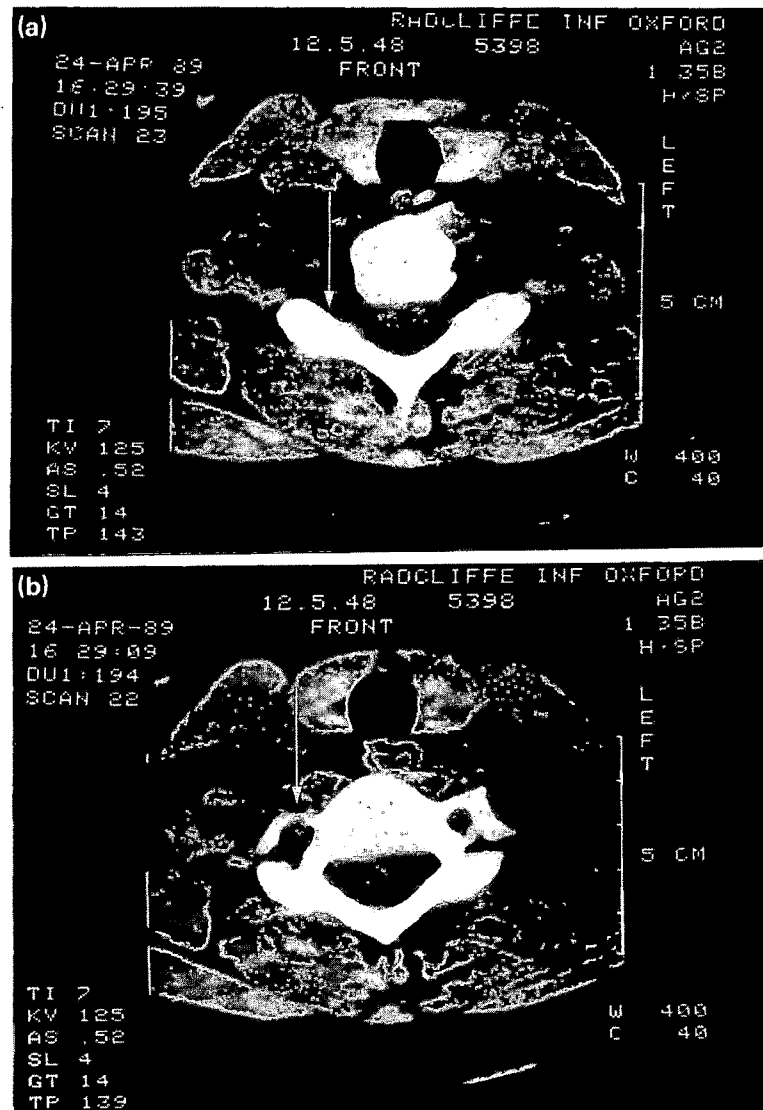


Fig. 1. Myelogram of the cervical spine.

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Accepted 4 April 1990.



Figs 2 (a and b). CT scans of the cervical spine at the level of C₆. The white arrows represent the position of the block needle. The distance between these contiguous horizontal 'slices' is 4 mm. Figure 2 (a) represents the presumed level at which the block was performed. The needle is seen lying within the spinal nerve sulcus, and if it had entered the neck a few millimetres cephalad (Fig. 2b) the CSF would have been protected by the transverse process of C₆.

Discussion

The anterior (paratracheal) approach to the stellate ganglion was described by many authors^{6,8-10} and appears to be the safest route to avoid serious complications including subarachnoid injection.^{3,6} Injection is usually performed at the level of C₆ since it was shown that the volume of local anaesthetic required to block the postganglionic sympathetic fibres at this level is less than at C₇.¹¹ There are three reported cases of subarachnoid injection by the paratracheal route in the literature; two are attributed to the patient coughing during the procedure^{1,2} and one to the opening up of tissue planes in the neck by previous injections.⁴

The potential for intradural injection can be seen if the anatomy of the cervical spine is considered. The usual bony landmark is the anterior tubercle of the transverse process of C₆ (the Chassaignac tubercle). The needle is introduced

perpendicular to the skin and advanced until it encounters the tubercle. However, if the point of entry is a few millimetres either cephalad or caudad to this landmark the advancing needle may first contact the bone of the superior articular process of the vertebra where it arises from the pedicle, i.e. *inside* the spinal nerve sulcus, and the needle may thus enter a dural cuff (Fig. 2). It is said that in this case resistance (from muscles extending between the transverse processes) will be encountered before hitting bone.^{12,13} This may be difficult to appreciate in clinical practice.

Subarachnoid injection is a serious and potentially fatal complication. It can be seen that the anatomy of the cervical spine is such that even using a 'safe' approach to the ganglion the dura is vulnerable to puncture. The case emphasises the need to pay attention to detail in performing these blocks. The importance of aspiration before injection should be stressed, although it should be noted that a negative aspiration test does not preclude

intradural injection if the needle is lying within a dural cuff.¹³ It is therefore suggested that aspiration be performed after each 2.5-ml increment of injected local anaesthetic to avoid placing dangerous quantities of drug intrathecally (or intravascularly). Such blocks, because of this and other potentially serious complications, should be performed in areas where full resuscitation equipment is available.³

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CASE REPORT

The use of diazepam in chloroquine poisoning

A. RAJAH

Summary

A 39-year-old patient was found to be unconscious after having taken 2.5 g of chloroquine. Treatment consisted mainly of gastric lavage and diazepam. Experimental and clinical evidence is presented to show that diazepam in varying doses significantly decreases the mortality rate.

Key words

Toxicity; chloroquine.

Hypnotics; benzodiazepines, diazepam.

The antimalarial drug chloroquine is the most severe and frequent cause of poisoning in Africa^{1,2} and the Far East.³ The mortality rate in the published studies ranges between 10 and 30% and is amongst the highest in clinical toxicology.⁴ The high mortality rate in chloroquine poisoning is related to close dose-dependent toxicity and to rapid onset of severe cardiac symptoms. The use of diazepam as an anti-arrhythmic agent in chloroquine poisoning has been known for some time but there does not appear to be any references to this in the British medical literature.

Case history

A 39-year-old, previously healthy but severely depressed, Caucasian woman was admitted 3 hours after a multiple overdose of chloroquine 2.5 g, maloprim 100 mg, some sudafed tablets (that contain paracetamol), and also some antismoking and slimming tablets. She was unconscious before admission.

On examination, she was noted to have peripheral and central cyanosis, tachypnoea with a rate of 28 breaths/minute. Her lung fields were clear. She had a pulse rate of 114 beats/minute and an arterial blood pressure of 100/60 mmHg. Her level of consciousness had improved at the time of being seen in the casualty department when she was found to be drowsy but responding to verbal commands. There was no other neurological deficit.

Investigations showed the full blood count, electrolytes and glucose to be within the normal range. Her arterial blood gases whilst breathing an F_{IO_2} 0.6 were: pH 7.45, P_{CO_2} 5.57 kPa, P_{O_2} 26.38 kPa, BE 5.7 mmol/litre and O_2 saturation of 99%. The blood chloroquine level determined subsequently was 6 μ mol/litre and the blood paracetamol

level 0.03 mmol/litre. Her methaemoglobin level was 30% on admission. The ECG confirmed the sinus tachycardia with a QRS duration of 0.10 seconds.

Gastric lavage was performed in the casualty department, after which she was admitted to the intensive care unit. She was given diazepam 20 mg intravenously initially with further doses to be given if warranted by her clinical conditions. The methaemoglobinaemia induced by maloprim was treated with methylene blue 1 mg/kg; the level dropped to 10% the next day. Her condition remained stable over the next 48 hours when she was transferred to the ward.

Discussion

Chloroquine has a very low margin of safety. The recommended dose for an adult is 600 mg or 10 mg/kg. It is considered by most authors that, for an adult, 1 to 1.5 g chloroquine (20 mg/kg) is a toxic dose and 2 g (30 mg/kg) may be lethal.⁵ A fatal outcome was reported after intravenous administration of 250 mg in a 42-year-old man.⁶ Our patient in this case report ingested chloroquine 2.5 g, which could have been lethal.

The clinical features of chloroquine poisoning are predominantly related to the cardiovascular system, although drowsiness leading to coma and ventilatory disturbances (apnoea, hyperventilation) may be seen. The cardiotoxicity of chloroquine is due to its quinidine-like action. Chloroquine has a negative inotropic action, inhibits spontaneous diastolic depolarisation, slows conduction, lengthens the effective refractory period and raises the electrical threshold. This results in depression of contractility, impairment of conductivity, decrease of excitability, but

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Accepted 23 April 1990.

with possible abnormal stimulus to re-entry mechanisms.⁷⁻⁹ However, cardiac arrest can also be the first manifestation of chloroquine overdose.¹⁰

Don Michael *et al.*¹¹ described a progression of ECG changes in dogs with experimental chloroquine overdosage. Sinus tachycardia preceded loss of voltage and widening of the QRS, followed by sinus bradycardia, ventricular tachycardia, ventricular fibrillation and finally asystole.

Various modes of treatment have been tried in order to increase its elimination from the body, but all have been found to be ineffective.¹²⁻¹⁴ The reason for the failure of the haemodialysis, haemoperfusion and peritoneal dialysis is probably because chloroquine rapidly becomes intracellular after administration. Early gastric lavage is essential and of considerable value.

Experimentally it was shown that pentobarbitone-diazepam anaesthetised dogs could survive after the administration of chloroquine 200 mg/kg.¹⁵ In another study¹⁶ the effects of diazepam administered intraperitoneally in rats immediately after an oral LD₅₀ dose of chloroquine (300 mg/kg) were studied. A significant decrease in mortality was observed at diazepam doses between 7.5 and 55 mg/kg, with an optimal effect at 20 mg/kg. In 1986, Riou *et al.*¹⁷ administered diazepam 2 mg/kg over 2 minutes and then 1 mg/kg/hour to pigs intoxicated by 50 mg/kg chloroquine and this significantly reduced the cardiotoxic effects.

Clinically the usefulness of diazepam in chloroquine poisoning was highlighted by Djelardje.¹⁸ He noted that in a patient who took chloroquine 5 g together with diazepam 500 mg no clinical symptoms of chloroquine toxicity were observed. The mortality rate from chloroquine poisoning was reduced by 10-fold by parenteral administration of diazepam. No clinical features of chloroquine toxicity were observed in three patients who had ingested chloroquine (2.2, 2.6 and 5 g) together with diazepam.¹⁹

Recovery after cardiac arrest only after intravenous injection of diazepam was reported in three cases of chloroquine poisoning.²⁰⁻²¹ Bouvier *et al.*²² reported three patients with severe chloroquine poisoning. In the first, shock and ventricular tachycardia responded to diazepam treatment but the patient died from ventricular tachycardia after 56 hours, 8 hours after diazepam withdrawal. The other two patients survived where the diazepam administration was continued up to the 10th day after intoxication. Nine cases of severe (chloroquine 6 g) poisoning were reported by Barriot *et al.*²³ Six patients not treated with diazepam died from cardiac arrest within 2 to 3 hours. Three patients treated immediately with diazepam and dobutamine recovered without severe cardiac disturbances.

Riou *et al.*²⁴ retrospectively selected a control group of 11 consecutive patients who had ingested more than chloroquine 5 g in a 2-year period. They then selected the following 11 consecutive patients who had taken more than chloroquine 5 g, to receive immediate mechanical ventilation and administration of diazepam and adrenaline. Ten of these patients, but only one control, survived. A report by Havens *et al.*²⁵ documents the survival of a 17-month-old child after ingestion of chloroquine 4 g. He developed ventricular fibrillation and cardiovascular collapse one hour after ingestion. He was treated with diazepam 2 mg/kg after resuscitation, followed by 0.25 mg/kg/hour for 4 days with no further cardiac abnormalities.

The mechanism of action of diazepam in chloroquine poisoning has been debated for some time. Koudogbo *et*

*al.*²⁶ noticed that when diazepam was administered to chloroquine-intoxicated rats, it increased chloroquine concentration in the blood, but decreased it in the heart muscle. The peripheral type of benzodiazepine binding sites (independent of the GABA receptor) was described in several animal tissues such as heart, lung, kidney, liver, uterus, adrenal cortex and blood cells. These were initially regarded as simple acceptors,²⁷ but this concept was recently challenged by the possible implications of peripheral benzodiazepine receptors in the anti-arrhythmic properties of diazepam.²⁸ Thus, the recent discovery of benzodiazepine receptors in human heart muscle would be in favour of competition of diazepam at cardiac chloroquine fixation sites.²¹

Diazepam dosage regimens vary according to different authors. It appears difficult to standardise diazepam treatment and the dosage and duration of treatment has to be individually adapted to the severity. Dosage regimens vary from 0.5–1 mg/kg with a possible continuous infusion (5 to 10 mg/hour) over the next 48 hours. Tracheal intubation and mechanical ventilation should be instituted to counter respiratory depression.⁴

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CASE REPORT

Caesarean section in a patient with haemoglobin SC disease and a phaeochromocytoma

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Summary

The anaesthetic management of a patient with haemoglobin SC disease for lower segment Caesarean section and excision of a phaeochromocytoma is described. The patient was given a general anaesthetic for the surgical procedure after exchange transfusion had achieved an haemoglobin A concentration of greater than 50%. A live infant was delivered and a suprarenal phaeochromocytoma was excised during a 6.5 hour procedure. The patient's postoperative recovery was uneventful.

Key words

Surgery; phaeochromocytoma.

Blood; sickle cell disease.

We describe the anaesthetic management of a patient with haemoglobin SC disease who had simultaneous lower segment Caesarean section and excision of a phaeochromocytoma. The management of such a case has not been described previously.

Case history

A 32-year-old multiparous woman with a confirmed history of haemoglobin SC disease was admitted from the antenatal clinic at 35 weeks' gestation with hypertension and proteinuria. She complained of intermittent severe headaches of recent onset.

On the ward her arterial blood pressure was noted to be labile, and ranged from 110/60 to 190/115 mmHg. A phaeochromocytoma was suspected and the diagnosis was confirmed by finding raised urinary vanillyl mandelic acid ($> 40 \mu\text{mol}$ in 24 hours) and plasma noradrenaline (4900 pg/ml). An ultrasound scan demonstrated a 4-cm right adrenal mass. Treatment with phenoxybenzamine 10 mg twice daily was commenced. A case conference was held between anaesthetists, haematologists and obstetricians. It was decided to perform an exchange transfusion before Caesarean section and excision of the phaeochromocytoma.

She was admitted to the intensive therapy unit where

direct arterial and central venous pressure monitoring were established before exchange transfusion began. The results from sequential haemoglobin electrophoresis during exchange transfusion are shown in Table 1.

The next day she received a premedication of diazepam 5 mg given orally 2 hours before induction, and 0.3 M sodium citrate 30 ml immediately before induction. Monitoring consisted of continuous ECG, direct arterial and central venous pressures, core temperature, pulse oximetry, capnography, peripheral nerve stimulator and urinary output. After pre-oxygenation and application of cricoid pressure a rapid sequence induction was performed using thiopentone and suxamethonium. Phentolamine, in boluses of 1 to 2 mg, was given in response to the hypertension associated with laryngoscopy and intubation. Tubocurarine 30 mg was given to provide muscle relaxation. Anaesthesia was maintained with 50% nitrous oxide in oxygen supple-

Table 1. Results of haemoglobin electrophoresis during exchange transfusion.

	HbA	HbS	HbC
Before exchange transfusion	3.0%	54.0%	43.0%
After exchange of four units	30.1%	33.6%	33.2%
After exchange of eight units	55.3%	21.7%	23.0%

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Accepted 29 March 1990.

mented with 0.75% isoflurane. A glyceryl trinitrate infusion was used to keep the systolic arterial blood pressure below 150 mmHg. A live male infant was delivered by lower segment Caesarean section. Anaesthesia was then supplemented with fentanyl 5 µg/kg. Excision of the phaeochromocytoma took a further 6 hours. The systolic blood pressure was kept between 100 and 150 mmHg using a background glyceryl trinitrate infusion and boluses of labetalol and phentolamine as necessary. Total blood loss was 3 litres.

The residual neuromuscular blockade was reversed at the end of the operation and her trachea was extubated when she was awake in the head-down left lateral position. She returned to the intensive therapy unit and remained overnight breathing oxygen-enriched air. She was discharged to the postnatal ward the next day. She suffered no post-operative complications and was discharged 10 days later.

Discussion

Phaeochromocytoma in pregnancy is rare. A recent review identified only 128 cases in the world literature.¹ The symptoms and signs of this condition are similar in the pregnant and nonpregnant patient, but the diagnosis is often missed because of its rarity and because the clinical picture may mimic that of pre-eclampsia. Potentially fatal hypertensive crises may be precipitated by vaginal delivery, uterine contractions, anaesthesia or even vigorous fetal movements.² Maternal mortality associated with phaeochromocytoma was 48% before 1969, decreased to 26% in the period 1969 to 1979,³ and was further reduced to 17% in the period 1979 to 1987.⁴ Antepartum diagnosis reduces both maternal and fetal mortality.²

The association of sickling disease and pregnancy also has an increased maternal and fetal mortality.⁵ Vaso-occlusive sickle cell crisis is the most common maternal complication noted in pregnancy associated with sickle cell disease.⁶ Meticulous medical care can significantly reduce pregnancy failure and complications. Exchange transfusion has been recommended for prevention of sickle crises in pregnancy;⁷ the aim is to achieve a proportion of haemoglobin A of 50 to 60%.⁸

In planning the anaesthetic management of such a high-risk patient it was decided that the patient should be managed by a consultant anaesthetist (J.C.R.) experienced in both obstetric anaesthesia and anaesthesia for patients with phaeochromocytoma. Exchange transfusion was performed before operation to minimise the risk of sickle crisis. It was also decided that she should be managed on the intensive therapy unit during the exchange transfusion and for the immediate period after operation.

The provision of regional anaesthesia was considered. However, it was rejected for a number of reasons. It is unlikely that the hypertensive response to noradrenaline release from the phaeochromocytoma would have been completely obtunded by regional anaesthesia. It has been suggested⁹ that regional anaesthesia, by interrupting the nerve supply to the adrenal gland, should obtund neurogenic stimulation of the tumour. Spinal and epidural anaesthesia to midthoracic dermatomal levels blocks preganglionic sympathetic fibres that supply blood vessels in the lower limbs and splanchnic circulation. However, noradrenaline released from a phaeochromocytoma by inadvertent surgical manipulation would be expected to act

directly on alpha adrenoceptors in the blood vessels themselves and therefore the risk of hypertension would not be avoided. It would have been difficult to provide an adequate anaesthetic block for the excision of the phaeochromocytoma, and therefore this approach would have exposed the patient to the risks of both regional and general anaesthesia. The patient was also most reluctant to accept a regional anaesthetic.

The successful use of epidural anaesthesia for Caesarean section in two patients with phaeochromocytoma was reported.⁹ However, both patients had had previous phaeochromocytoma excision; the site of their recurrence had not been radiologically demonstrated before operation and the tumours were not removed at the time of Caesarean section. In the above report, as in another of simultaneous Caesarean section and phaeochromocytoma removal under general anaesthesia,¹⁰ the patients were suffering from no other complicating medical conditions.

Our choice of phentolamine, glyceryl trinitrate and labetalol was based largely upon experience with these agents and personal preference. Sodium nitroprusside, which is recommended in the management of hypertension in patients undergoing phaeochromocytoma excision,¹¹ was avoided because it is thought to reduce uterine blood flow substantially,¹² and evidence from studies on fetal lambs suggests that acidosis and cyanide accumulation occur to a greater extent than in the mother.¹³

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An evaluation of the Level 1 blood warmer series

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Summary

The Level 1 blood warmer series comprises three infusion sets and two blood warmers of different power outputs. All systems were found to be extremely efficient, with the larger 500 series capable of warming the equivalent of 80 units of blood an hour almost to body temperature.

Key words

Equipment; blood warmer.

Hypothermia can be a serious problem after massive blood transfusion and the need for maintenance of normothermia under these circumstances is firmly established. The major problem associated with conventional blood warmers are the limitations in flow because of the resistance of the tubing in the apparatus and the very poor thermal conductivity of the plastic of the infusion sets. As a result these warmers become very inefficient at flows greater than about 150 ml/minute.

The Level 1 series* is a new type of blood warmer that has recently become available in this country, and we report an investigation of it using a previously described method.¹

Apparatus

The Level 1 series consists of two components, a hardware system and a disposable giving set.

Hardware

The warming unit is attached to an infusion stand and heats water to 40°C. This is then circulated through an aluminium single counter current heat exchanger (Fig. 1) which is an integral part of the infusion sets and is attached to the infusion stand. The latter also incorporates an adjustable height pole. Two independent circuits continuously control and monitor the water temperature and will

alarm and shut the system down in the event of over-heating. Two models are available.

250 model (Fig. 2a). This unit has a warming capacity of 600 watts and is attached to the vertical limb of the infusion stand. An indicator light shows that the system is operational and warning lights indicate if the disposables are not properly set up, if water needs to be added to the unit and if the temperature exceeds 41°C. There is only one on/off switch. The assembled height with the intravenous pole varies from 135 to 229 cm and the assembled dry weight is 10 kg.

500 model (Fig. 2b). This is a larger unit than the 250 model with a warming capacity of 1000 watts. It is attached to the base of the infusion stand; the indicator lights are as for the smaller apparatus and again there is only one switch. The height with the intravenous pole varies from 163 to 234 cm and its assembled dry weight is 25 kg.

A small compressor unit that can be attached to the base of the vertical portion of the infusion stand is also available. This can be used automatically to inflate either a 500- to 1000-ml pressor infuser attached to the top of the stand.

Disposable giving sets

There are three different pre-assembled and sterile disposable giving sets; the D50, D100 and D300 are complete and consist of ports for entry into the fluid/blood bag (two for the D50 and D100 and three for the D300), heat exchanger, air eliminator and tubing from the latter to the intravenous cannula. Water at 40°C circulates through the heat

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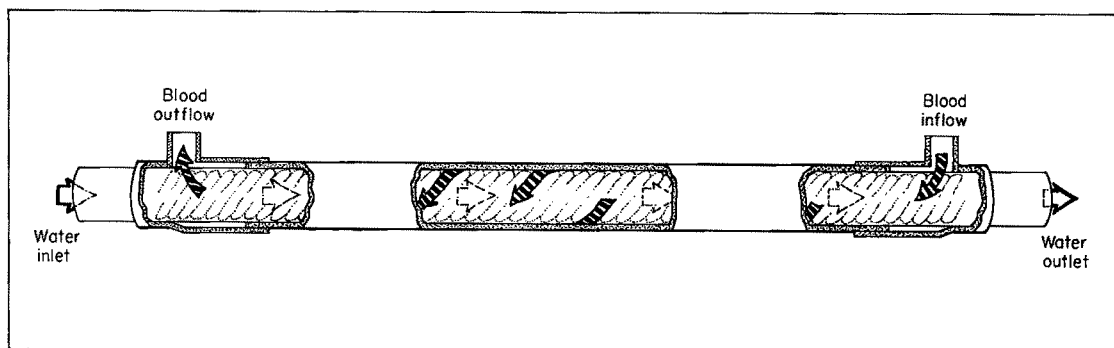


Fig. 1. Diagram of aluminium single counter current heat exchanger.

exchanger and the fluid warmed in the latter is gently mixed to ensure effective heat exchange; fluid flow is not restricted since the diameter within the heat exchanger is approximately the same as the internal diameter of the giving set. Each set can be used with either of the warming units.

D300. The system accommodates the largest flows and is recommended for liver surgery and transplantation. The tubing has an internal diameter of 4.8 mm along its entire length and a priming volume of 90 ml.

D100 is recommended in cases of major trauma. Its internal diameter is 4.8 mm to the end of the heat

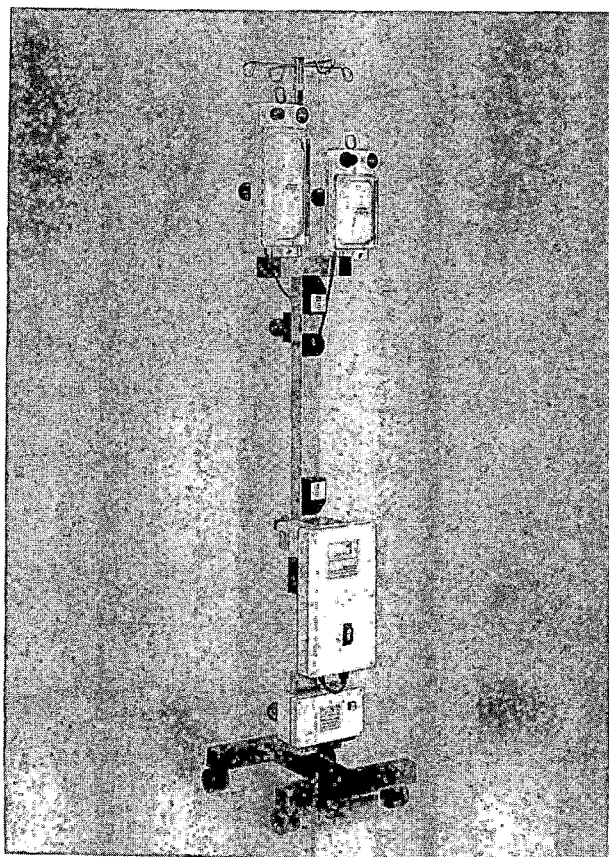


Fig. 2a. The 250 model apparatus. The warming unit is attached to the vertical portion of the infusion stand, beneath which is the small compressor unit.

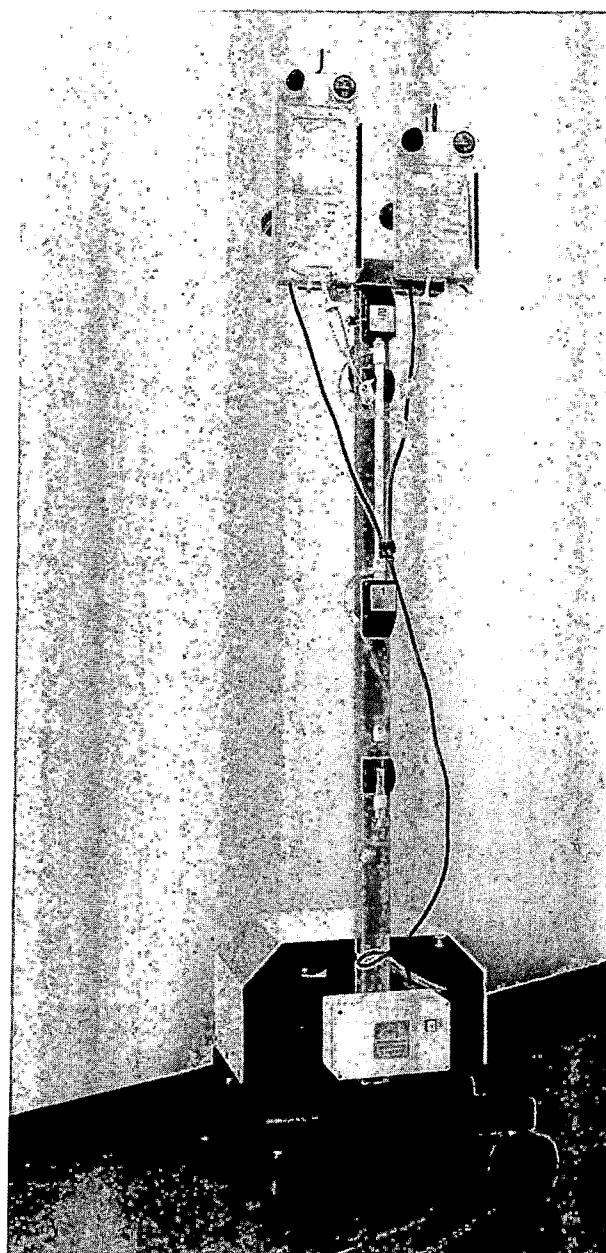


Fig. 2b. The 500 model with the D300 infusion set attached. The warming unit is attached to the base of the infusion stand and the small compressor unit to the bottom of the vertical portion.

exchanger and from there to the patient is 3.3 mm. It has a priming volume of 65 ml.

D50 is recommended for most other procedures. It has an internal diameter of 3.3 mm throughout its entire length and a priming volume of 50 ml.

All the sets employ an air eliminator capable of venting up to 400 ml/minute of air from the system. It also contains a 170-micron screen filter. The manufacturers recommend that these are changed every 3 hours to ensure efficiency of the overall system. The average time for priming the sets is less than 30 seconds.

The disposable giving set is interfaced with the pole assembly as shown in Fig. 2b. The air exchanger with the 250 model is attached to the heating unit and with the 500 model to the infusion pole. A low voltage interlock circuit with alarm is incorporated to confirm correct attachment of the giving set.

Methods

The infusion sets were connected to one-litre bags of normal saline refrigerated for 24 hours, and the inlet temperature of the saline varied from 1.8 to 4.0°C. The fluid issuing from the end of the giving set was collected in a closed container and the temperature measured at the entrance to the container. The thermistor used (American Edwards Laboratories) was calibrated against a National Physical Laboratories calibrated thermometer and was not incorporated into the tubing so as not to restrict the fluid flow.

Temperature was measured at flows up to 600 ml/minute using the D50, D100 and D300 disposable sets with the 250 and 500 units. The different flows were obtained by using gravity and pressures of 150 mmHg and 300 mmHg applied to pressor infusers. The distance from the heat exchanger to the point of temperature measurement varied according to the disposable set used; it was 180 cm for the D50, 208 cm for the D100, 286 cm for the D300 and 150 cm for the Fenwal. The latter was tested since it was the routine blood warmer in use in this department. Each test was repeated several times, but there was no measurable difference between them.

Results

250 model. The results obtained using this model with the D50 and D100 giving sets are shown in Figure 3, together with those of the Fenwal warmer. The issuing temperature decreased with increasing flow with all systems. With flows up to 250 ml/minute the D50 and D100 systems consistently warmed the fluid to over 34°C and to 32.5°C or above at 300 ml/minute. The Fenwal was only able to maintain an outlet temperature of 32°C or above at flows just over 100 ml/minute.

The maximum flow that could be achieved using an 8.5 gauge cannula attached to the giving sets, with a pressure of 300 mmHg with the D50 and D100 sets was 525 and 555 ml/minute respectively with an outlet temperature of 27.6°C.

500 model. The results obtained with the D100 and D300 infusion sets are shown in Figure 4. A temperature of 34°C or above was maintained with both sets at flows up to 600 ml/minute; the D100 performed slightly better. A temperature of 35°C was achieved with the latter at almost 700 ml/minute. Again, the Fenwal was unable to achieve a satisfactory temperature at flows over 125 ml/minute.

The maximum flow that could be obtained with the D300 unit with an 8.5-gauge cannula and a pressure of 300 mmHg was 1200 ml/minute; the issuing temperature at this flow was 27.2°C.

The water bath reaches operating temperature (40°C) in 2 minutes.

Discussion

Most commercially available blood warmers were, until recently, unable to maintain an outflow temperature above 32°C at flows greater than 150 ml/minute. The inefficient heat exchange resulted from the extremely poor thermal conductivity of plastic, which is in fact an insulator. The warmers involved either a long length of plastic tubing immersed in warm water or passage of blood through a plastic bag placed between heated plates. The air interface in the latter also acted as a filter. The length of tubing involved increased resistance and limited flow.

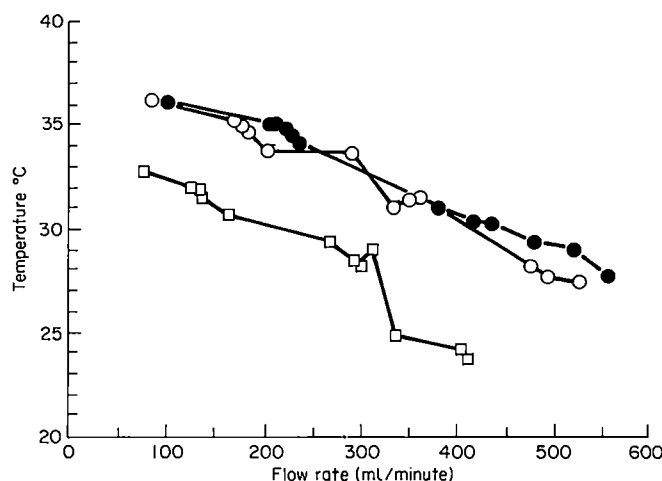


Fig. 3. Relationship between flow rate and issuing temperature using the Level 250 warmer, the D50 and D100 giving sets and the Fenwal warmer. —□—, Fenwal; —○—, 250/D50; —●—, D250/D100.

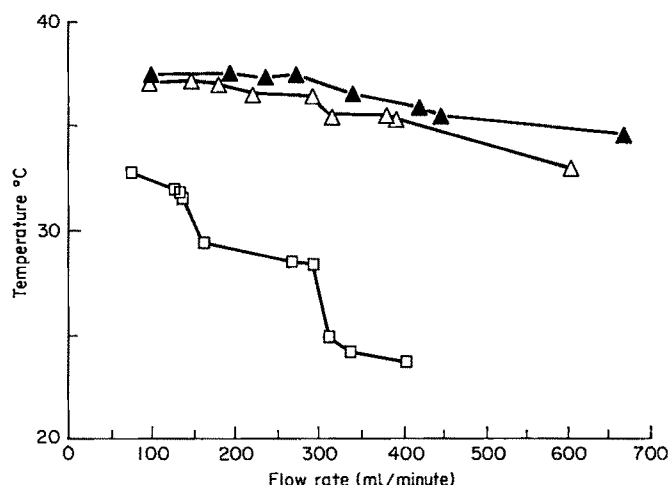


Fig. 4. Relationship between flow rate and issuing temperature using the Level 500 warmer with the D100 and D300 giving sets. Results from the Fenwal warmer are also shown. —□—, Fenwal; —▲—, 500/D100; —△—, 500/D300.

The Level 1 system introduces a new concept to blood warming. Water heated and kept at 40°C is pumped countercurrent through a tube-in-tube heat exchanger. The inner tube is thermally conductive anodized aluminium that has about 1000 times the heat transfer efficiency of plastic. The system, as a result, is capable of warming cold fluids to temperatures close to body temperature at high flows. The smaller 250 model consistently warmed saline at 4°C and at flows of 250 ml/minute to above 34°C. This is the equivalent of transfusing 33 units of blood per hour. The larger 500 model was even more efficient.

Russell² has calculated that an energy output of 310 watts is necessary to ensure that blood at 4°C is warmed to 32°C or above at a flow of 150 ml/minute. The power output of the 250 model (600 watts) and 500 model (1000 watts) means that such temperatures are well within the range of both these systems. The efficiency of the heating system is attested by the fact that temperature above 32°C can be obtained from the 250 unit at double this flow rate, whereas this is not so with the Hetotherm and Fenwal blood warmers that have similar power outputs.¹ The Fenwal did not perform quite as well in the present study compared to that reported previously because of the greater distance from the site of emergence from the blood warmer and the point of temperature measurement.

Russell's specifications² for blood warming apparatus were laid down in 1974, but there has been a marked increase in liver surgery since then and consequently massive blood transfusion. Provision of blood at a temperature above 32°C at flows up to 150 ml/minute is no longer tenable with the rates of transfusion required in this type of surgery, but it is essential that body temperature be maintained in situations of massive blood loss. The mortality in trauma victims of hypothermic patients was significantly higher than those who remained warm and was 40% if the core temperature decreased below 34°C, 69% if below

33°C, but no patient survived if core temperature decreased below 32°C.³ The Level 1 500 model was capable of maintaining a temperature output close to body temperature at flows of 600 ml/minute, which is equivalent to a transfusion rate of 80 units/hour.

The Level 1 system also met the criteria for allowing high flow rates, a low priming volume and compact design, and we found the disposable sets could be easily and rapidly set up and primed. It has also been shown that prolonged exposure to the 40°C heat exchanger (flow rate equivalent to transfusion at a rate of one unit per 4 hours) does not cause red cell damage.⁴

In conclusion, the Level 1 blood warming systems have been found to be extremely efficient, particularly the 500 model. Our clinical experience to date has been limited and no case has required massive transfusion. The major drawback in this country will probably be cost, but its effectiveness is undisputed and the cost considerably less at the moment than other similar available equipment.

Acknowledgments

We thank Mr R. Penberthy of Penco Medical Ltd for supplying the Level 1 warming systems and Mrs S. Richens for secretarial assistance.

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Forum

Embolism during Caesarean section

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Summary

We investigated the occurrence of gas embolism during Caesarean section using a Doppler ultrasound probe and found that it occurs between uterine incision and delivery. Embolism is less common during general anaesthesia than has been reported during regional anaesthesia. Both ruptured membranes and a protracted uterine incision to delivery interval predispose to embolism.

Key words

Surgery; Caesarean section.

Complications; air embolism.

Air embolism associated with chest pain during Caesarean section under regional anaesthesia was detected in 50% of patients.¹ We studied air embolism during general anaesthesia for both elective and emergency Caesarean section, to determine its incidence, timing in relation to surgery, predisposing factors and any sequelae.

Method

A Doppler ultrasonic probe (Parks medical 821), attached parasternally over the right fourth intercostal space, was connected to one channel of a stereo tape recorder. The recording was marked at skin incision, uterine incision, delivery of the baby, the beginning of uterine suturing and twice after skin closure. All the tapes were subsequently analysed and embolism diagnosed when the characteristic sound (typically a harsh blowing or rushing sound) persisted for 10 minutes or more. The indications for surgery are given in Table 1.

A standardised anaesthetic was used in accordance with the prevailing policy. Mist. magnesium trisilicate 15 or 30 ml was administered before operation; an indwelling intravenous cannula was inserted under local anaesthesia, then hyoscine 0.2 mg was given intravenously, followed by pre-oxygenation, application of cricoid pressure and induction of anaesthesia with thiopentone 5 mg/kg. Tracheal intubation was facilitated by suxamethonium 1.5 mg/kg and paralysis maintained with a suxamethonium infusion. The patient's lungs were ventilated to normocapnia with 50% N₂O in oxygen, supplemented with 0.5% halothane, 0.5% isoflurane or 0.8% enflurane. Monitoring consisted of noninvasive blood pressure, ECG, end-tidal CO₂ and the Doppler. However, the end-tidal CO₂ results were not recorded.

The patient's age, parity, gravidity, indication for Caesarean section, uterine incision to delivery time and whether the membranes were ruptured were also noted.

Six of the tapes were uninterpretable and disregarded.

Great care was taken to ensure that artefacts (particularly those caused by fundal pressure) were excluded.

Results

All embolic phenomena occurred between uterine incision and delivery of the baby. Embolism was detected in two of the nine (22%) elective cases, and the Doppler changes lasted from 11 to 20 (mean 15) minutes.

Embolism occurred in 16 of the 39 emergency cases. It occurred in one of the five patients whose membranes were intact and in 15 of the 35 (43%) whose membranes were ruptured. These changes lasted from 14 to 28 (mean 21) minutes. There was a significant difference in the incidence of embolism in relation to membrane integrity (Table 2). Only three of 17 (18%) mothers with intact membranes developed Doppler evidence of embolism, compared to 15 out of the 32 (47%) mothers in whom the membranes were ruptured. Embolism was also more likely where the uterine incision to delivery was over 30 seconds.

There was no correlation between embolism and age, gravidity, parity or the indication for operation. The duration of Doppler change was not statistically significant

Table 1. Indications for Caesarean section.

<i>Elective</i>	
Pre-eclamptic toxemia	1
Breech presentation	2
Cephalopelvic disproportion	6
<i>Emergency</i>	
Fetal distress	17
Failure to progress	12
Antepartum haemorrhage	4
Pre-eclamptic toxemia	4
Intra-uterine growth retardation	1
Prolonged ruptured membranes	1

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Accepted 11 July 1989.

Table 2. Incidence of embolism.

Uterine incision to delivery time	No embolism	Embolism
Less than 30 seconds	11	4
More than 30 seconds	20	14
Membrane integrity		
Membranes intact	14	3
Membranes ruptured	17	15

There is a significant relationship between the incidence of embolism and uterine incision to delivery time of over 30 seconds ($p < 0.05$) Wilcoxon's rank sum test. There is also a significant increase in the incidence of embolism in patients with ruptured membranes when compared to those with intact membranes ($p < 0.05$) Chi-squared test.

between groups; no changes in end-tidal CO_2 or blood pressure were noted by the anaesthetists.

Discussion

Malinow *et al.*¹ found an incidence of embolism of 50% that correlated well with complaints of chest pain in patients undergoing Caesarean section under regional anaesthesia.

The overall incidence in this series under general anaesthesia was 37%; embolism occurred between uterine incision and delivery, and possibly the venodilatation accompanying regional anaesthesia makes embolism more likely. IPPV increases central venous pressure and might protect against embolism.

Anaesthesia, 1990, Volume 45, pages 965–968

Anaesthesia in the field Spontaneous ventilation—a new technique

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Summary

In recent years the British Army has used the Triservice Anaesthetic Apparatus in the field. Trichloroethylene is no longer manufactured in the United Kingdom and halothane is not recommended for closely repeated anaesthetics. A method based on existing equipment is described for patients breathing spontaneously. A background infusion of ketamine, midazolam and alfentanil supplements the inhalation of isoflurane in oxygen-enriched air.

Key words:

*Anaesthetic techniques; drawover.
Anaesthetics, volatile; isoflurane.*

All anaesthetists who work with patients who require surgery for injuries by gunshot or explosions are aware that the overriding principle of such surgery is that of delayed primary suture. Consequently, most of these casualties

Aspiration of amniotic material from the maternal pulmonary artery was described in an asymptomatic mother,² but we believe that Doppler changes that persist for more than 10 minutes are from gas bubbles trapped in the right atrium, and that solid emboli would pass into the pulmonary artery and the Doppler signal would fade.

The embolic events were not associated with detectable sequelae, but it was shown that in the awake patient, embolism leads to complaints of chest pain.¹ The frequency of embolism during Caesarean section is important, since the development of a serious embolus is determined by dosage and rate of entry of the gas. Massive air embolism has been described in Caesarean section.³

Acknowledgments

We thank the late Dr J.S. Crawford for his encouragement. We also thank the staff of the Birmingham Maternity Hospital for their cooperation.

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require more than one anaesthetic. All patients who suffer such injuries must be assumed to have a full stomach at the first operation and require muscular paralysis, tracheal intubation, artificial ventilation and light anaesthesia. A

technique using a facemask, with the patient breathing spontaneously, may be a more attractive alternative for subsequent operations.

Satisfactory anaesthesia under field conditions can be achieved using the Tri-Service Apparatus (TSA) with halothane and trichloroethylene. This technique has been well tried and documented, and has been the basis of British military anaesthesia for the past 15 years.¹ However, halothane is hepatotoxic and closely repeated halothane anaesthetics are not acceptable in modern practice.² The manufacture of trichloroethylene in this country has ceased and it is no longer possible to purchase trichloroethylene passed for medical use in peace time in the UK.

Both enflurane³ and isoflurane⁴ are recommended for use with the TSA. Unfortunately enflurane has a high minimum alveolar concentration (MAC) and cannot be administered as a single agent at sufficient concentration from one Oxford Miniature Vaporizer (OMV) to induce and maintain an adequate depth of anaesthesia. Isoflurane, without nitrous oxide, can be difficult to use because of its irritant effect on the trachea. It is also considerably more expensive than the other available inhalational agents.

We have attempted to develop an anaesthetic technique based on the total intravenous method which we have described previously.⁵ It must be simple so that it can be used in a hazardous environment with the minimum of equipment. Satisfactory operating conditions must be provided and the patients must awake and be able to maintain a safe airway as soon as possible after operation.

The method of total intravenous anaesthesia in the field that uses ketamine, midazolam and vecuronium was detailed in an earlier report.⁵ Unfortunately it has not proved possible to use a total intravenous technique based on the same drugs for patients breathing spontaneously and still obtain a short recovery time. Consequently, we have developed a technique which comprises an intravenous background anaesthetic of ketamine, midazolam and alfentanil, supplemented with minimal concentrations of isoflurane from a drawover vaporizer. We elected to use isoflurane so that the technique would be practical in peace time and could be used in disaster or limited warfare situations. We consider that halothane would be simpler to use and would be the anaesthetic of choice in the event of major conflict. We compared this new method with the traditional TSA technique using halothane and trichloroethylene.

Methods

The patients who took part gave informed consent to participate in this trial. They presented for body surface surgery that was likely to last 20–45 minutes. They were between the ages of 16 and 55 years, ASA grades 1 or 2 and free from hypertension, ischaemic heart disease, cerebrovascular disease or psychiatric illness. Those undergoing drug therapy that might modify the response to the anaesthetic agents were excluded from the trial. Sixty patients were admitted to the trial and allocated randomly into one of two groups.

All the patients were premedicated with temazepam 20 mg orally one hour before operation. Anaesthesia was induced with midazolam 0.07 mg/kg, alfentanil 5 µg/kg and ketamine 2 mg/kg in patients in group 1. A continuous intravenous infusion was then started using an electrically (mains or battery) driven syringe pump (Advanced Medical Devices AMD PS6050) at the following rates: midazolam 50 µg/kg/hour; ketamine 2 mg/kg/hour; and alfentanil 10 µg/kg/hour. This mixture is obtained by mixing midazolam 5 mg, ketamine 200 mg and alfentanil 1 mg with dilution to 50 ml with normal saline. A convenient *aide memoire* to the

rate of administration of this mixture is:

$$\frac{\text{body weight (kg)}}{2} = \text{infusion rate (ml/hour)}$$

The patient was allowed to breathe isoflurane from an Ohmeda PAC (Portable Anaesthetic Case) drawover vaporizer;⁶ the inspired air was supplemented by oxygen 1 litre/minute. The isoflurane concentration was altered as necessary during the operation depending on the patient's responses to surgery, and administration of isoflurane was discontinued about 10–15 minutes before the end of the procedure. Unfortunately, in several cases, it proved impossible to predetermine the end of surgery and this affected the recorded recovery times adversely.

Anaesthesia was induced with thiopentone 4–6 mg/kg in patients in group 2. The facemask was then applied and the patient breathed halothane and trichloroethylene from the TSA. The inspired air was supplemented by oxygen 1 litre/minute. The concentrations of both volatile agents were altered as necessary throughout the operation.

Monitoring in both groups consisted of electrocardiography (ECG), indirect blood pressure measurement (Dinamap), pulse oximetry, respiratory rate, end-tidal carbon dioxide concentration and inspired oxygen concentration. The isoflurane concentration was monitored in group 1 using a Capnomac (Vickers Datex) but it was not possible to measure halothane and trichloroethylene concentrations because the two agents were used concurrently. The settings on the OMVs were recorded.

Observations made during the operation included a subjective assessment of the quality of anaesthesia such as movement, coughing and laryngeal spasm. The recorded cardiorespiratory variables were used to assess depth of anaesthesia.

Postoperative observations were made in the recovery room and 24 hours later. These observations included: time of opening of eyes either spontaneously or to command; time to give name and ward number; behaviour in the recovery room; incidence of dreaming; and the incidences of nausea and vomiting.

Results

The groups of patients were comparable in age and weight but there were more males in group 2 (Table 1). No ECG disturbances were noted in any patient in group 1; one patient in group 2 developed a junctional rhythm. Systolic blood pressure remained stable after induction and during maintenance in group 1. There was a decrease in systolic blood pressure of more than 10 mmHg after induction in only a few patients; a slight decrease in diastolic blood pressure of less than 10 mmHg occurred after induction but this stabilised at 5 minutes. There was a small decrease in both systolic and diastolic blood pressures of about 10 mmHg in patients in group 2, but these stabilised at 5 minutes. The maximum decrease in systolic pressure was 28 mmHg (from 130 mmHg). Heart rate in both groups was very similar.

Table 1. Demographic data.

	Group 1	Group 2
<i>n</i>	30	30
Male: female	20:10	23:7
Age; years, mean (SD) range	29.7 (11.5) 16–55	24.7 (7.3) 16–43
Weight; kg, mean (SD) range	74.7 (12.6) 47–97	71.5 (8.5) 50–95

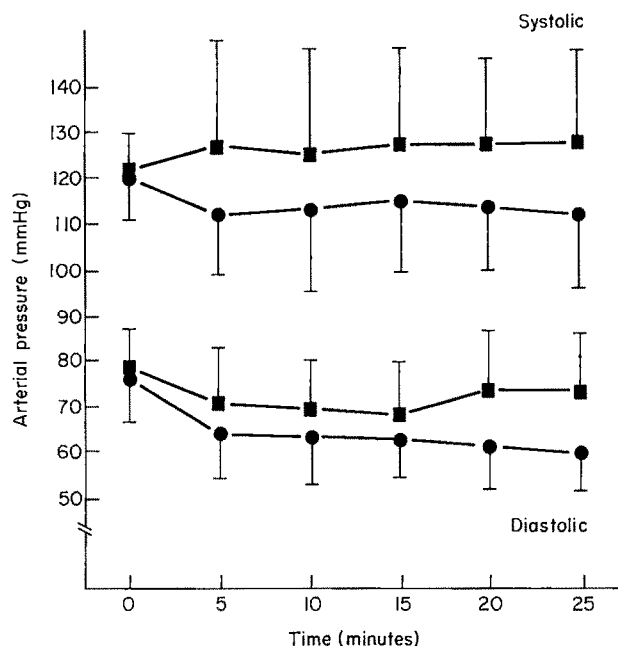


Fig. 1. Systolic and diastolic blood pressure in group 1 (■) and group 2 (●). Bars indicate SD.

There were no differences in oxygen saturation or inspired oxygen concentration between the two groups. There were minimal changes in respiratory rate and end-tidal carbon dioxide tension during the procedure in patients in group 1. However, end-tidal CO_2 decreased in group 2 between 5 and 10 minutes after induction, and then reached a plateau at approximately 4.6 kPa. The respiratory rate, as might be expected with the use of trichloroethylene, increased throughout the procedure.

We used higher concentrations of volatile agent initially, and a lower concentration for maintenance, in both groups. Isoflurane concentration was decreased rapidly in group 1 for the first 15 minutes, and subsequently end-tidal concentration was approximately 0.75%. The halothane and trichloroethylene vaporizer settings were decreased more slowly to a maintenance plateau.

The operation and recovery times are shown in Table 2. The mean (SD) operation time in group 1 was 14.7 (7.7) minutes with a mean recovery time of 30.9 (13.9) minutes.

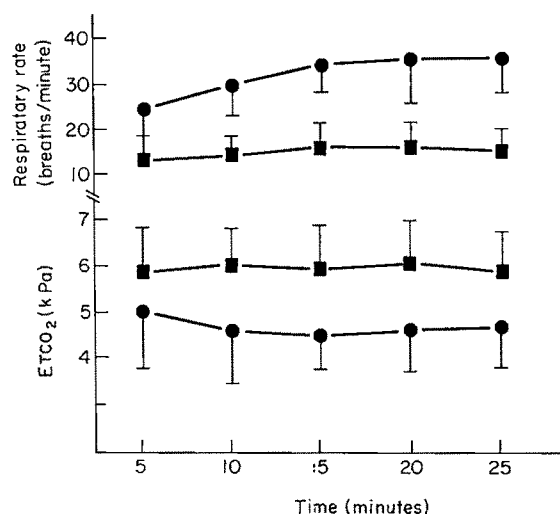


Fig. 2. Respiratory rate and end-tidal CO_2 concentration (ETCO_2) in group 1 (■) and group 2 (●). Bars indicate SD.

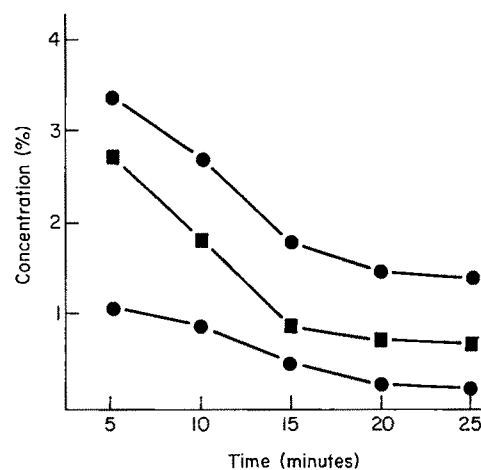


Fig. 3. End-tidal concentration of isoflurane in group 1 (■) and inspired vaporizer settings of halothane (upper) and trichloroethylene (lower) in group 2 (●).

The mean operation time in group 2 was 25.5 (12.8) minutes with a mean recovery time of 35.8 (14.4) minutes.

No patient in either group displayed grossly abnormal behaviour in the recovery room. Some patients stayed in the recovery room for a relatively prolonged period because they were thought by the nurses to be in a dissociated state.

The incidences of dreaming and of vomiting are shown in Table 3. Three patients in group 1 and five patients in group 2 vomited. Three patients in group 1 and two patients in group 2 had dreams, none of which was unpleasant. Ninety-three percent of patients in group 1 found the technique acceptable compared with 90% in group 2.

Discussion

We set out to develop an anaesthetic technique for second and subsequent anaesthetics for operations in the field. This was necessary because of the unacceptability of halothane and the unavailability of trichloroethylene. Trichloroethylene may be obtainable in some parts of the world but we require a drug that is manufactured in the UK and that can be used in routine day-to-day practice in this country. A hazardous environment is not the place to use a drug for the first time. The British Army anaesthetists have found the TSA simple to use and we have a stock of OMV 50 vaporizers. This technique allows the with-

Table 2. Durations of operation and recovery. Data are presented as mean (SD) and range.

	Group 1	Group 2
Duration of operation; minutes	14.7 (7.7) 3-33	25.5 (12.8) 6-53
Duration of recovery; minutes	30.9 (13.9) 10-68	35.8 (14.4) 11-66

Table 3. Postoperative vomiting, dreaming and acceptability; numbers of patients.

	Group 1	Group 2
n	30	30
Vomiting	3	5
Dreaming	3	2
Acceptability (%)	28	27

drawal of one vaporizer and the addition of a syringe pump. The AMD PS6050 pump can be driven by 5 LR14 batteries ('C' cells) for approximately 100 hours, and is light, robust and easy to use.

This method of anaesthesia is relatively simple and produces excellent operating conditions with minimal familiarity. The technique does need to be practised, but so does any other drawover method of anaesthesia. Most importantly, the drugs used caused minimal cardiorespiratory changes, although they were used in ASA 1 or 2 patients in this study. Ketamine is more suitable than thiopentone in shocked or ill patients.

Duration of recovery was longer than expected, as a result of several factors. It was difficult to anticipate the end of some operations and the infusion was not discontinued early enough; this problem is less likely to arise when the operation is for the delayed primary suture of a wound. The duration of recovery was determined by recovery staff who were unfamiliar with ketamine anaesthesia. Therefore, although the patients had awoken and were able to maintain their airway, they were not returned to the ward because they were deemed to be in a mildly dissociated state. Isoflurane concentrations were reduced earlier as we became more familiar with the technique and recovery became more rapid. The mean recovery times were not improved by one patient who remained unconscious for more than 60 minutes despite being given doxapram and naloxone intravenously. He was given flumazenil 0.2 mg and awoke immediately. The patients in group 1 recovered more rapidly on average than those in group 2 despite these problems, but the operations in group 1 were shorter.

The new technique, using ketamine, midazolam and alfentanil intravenously and isoflurane by inhalation, proved to be at least as good as the well-tried technique employing the Triservice apparatus and conventional inhalation agents. The quality of anaesthesia was good, blood pressure remained stable, and the patients recovered in

comparable times. The benefit was that the tachypnoea due to trichloroethylene did not occur; tachypnoea can be relieved by administration of boluses of an opioid, but only at the expense of respiratory depression. We believe that this technique may be suitable in the field for patients with head injury. The amount of ketamine used is small and its effects on intra-ocular and intracranial pressures are modified by midazolam. Low concentrations of isoflurane do not cause an increase in cerebral blood flow provided that P_{aCO_2} is normal.⁷

We recommend this method as an acceptable means of anaesthetising a patient in a hazardous environment where sophisticated apparatus is not available and the patient can be allowed to breathe spontaneously.

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Anaesthesia, 1990, Volume 45, pages 968-971

Postoperative sore throat: topical hydrocortisone

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Summary

Forty patients undergoing tracheal intubation and controlled ventilation of the lungs for elective surgical procedures were studied. They were allocated randomly into one of two groups. The tracheal tubes used for group A patients were lubricated before insertion with water-soluble 1% hydrocortisone cream. Those for group B patients were lubricated with KY jelly. The incidence of postoperative sore throat was found to be significantly greater in group A. Topical 1% hydrocortisone cream is therefore ineffective in the prevention of postoperative sore throat.

Key words

Intubation, tracheal; complications.

Sore throat is a common complication of general anaesthesia. It may occur after mask anaesthesia, although tracheal intubation increases the incidence to a level as high as 90%.¹

Many factors contribute to this problem, but the association between sore throat and the area of contact between cuff and trachea has been clearly demonstrated.¹⁻⁶ This finding implies that irritation of the tracheal mucosa by the

Table 1. Surgical procedures undertaken.

	Group A (hydrocortisone) (n=20)	Group B (KY jelly) (n=20)
Stripping of varicose veins	4	4
Excision of breast lump	1	1
Inguinal hernia repair	1	2
Cholecystectomy		1
Manual dilatation of the anus		1
Percutaneous nephrolithotomy	2	
Arthroscopy	7	6
Hip arthroplasty		1
Excision of plantar spurs		1
Abdominal hysterectomy		2
Reversal of sterilisation	2	
Laparoscopy (dye tests)	3	1

tracheal tube cuff is an important aetiological factor in postoperative sore throat.

Disappointing results obtained using topical local anaesthetic agents to prevent postoperative sore throat are therefore not surprising. Such agents may limit potential damage to the tracheal mucosa by suppressing coughing or bucking on the tube, but have no intrinsic anti-inflammatory action. Out of six studies^{1,3,7-10} only one⁹ has shown a clear benefit, and in two studies^{1,3} local anaesthetic agents were associated with an *increased* incidence of sore throat.

The potential benefit of specific anti-inflammatory agents for the prevention of postoperative sore throat was suggested by an early study by Hamelberg.¹¹ Topical corticosteroids were used in this study in combination with local anaesthetic agents.

The aim of the present study is to assess the effect of topical hydrocortisone 1% on the incidence of sore throat after tracheal intubation for general anaesthesia.

Patients and methods

The study was conducted on 40 adult patients aged 18–74 years, ASA grades 1 or 2, and approval for the study was obtained from the local ethics committee. Patients selected were those about to undergo elective surgery for general, orthopaedic, gynaecological or urological procedures which would require tracheal intubation and controlled ventilation of the lungs. Patients undergoing surgery to the head and neck, or procedures that necessitated passage of a nasogastric tube were excluded.

Informed consent was obtained from the patients, who were randomly allocated into one of two groups. The patients' tracheal tubes were prepared before arrival in the anaesthetic room. Disposable PVC Portex 'Theatre' tubes were used for all patients (8.0 mm internal diameter for women and 9.0 mm internal diameter for men). For group A patients 2–3 ml water-soluble 1% hydrocortisone

cream was lightly smeared around the outside of the tube from the tip to 5 cm above the cuff. The same volume of 'KY' jelly was applied in the same way for group B patients.

A standard anaesthetic technique was used. Oral temazepam (10–20 mg) and metoclopramide (10 mg) premedication was administered one hour before operation. A sleep dose of thiopentone was used for induction of anaesthesia and alcuronium (0.3 mg/kg) was given for neuromuscular block. The patients' lungs were ventilated to normocapnia with 66% nitrous oxide and 33% oxygen supplemented with 1–2% enflurane. Papaveretum (0.1–0.3 mg/kg) was given for analgesia if required. Tracheal intubation was not attempted until 3 minutes after administration of the alcuronium.

The cuff of the tracheal tube was inflated, after insertion to the minimum pressure required to prevent gas leak on positive pressure ventilation. This pressure was recorded, and further recordings were made at half-hourly intervals thereafter.

The neuromuscular block was reversed with neostigmine and atropine on completion of the surgical procedure. The tracheal tubes were removed from the patients who were in the left lateral position once spontaneous ventilation had resumed, but before return of the cough reflex.

The patients, who were unaware of which lubricant had been used, were all interviewed in a standard fashion on the day after surgery (16–24 hours after the procedure). They were asked directly if they had experienced any sore throat, if so, they were asked to grade it as mild, moderate, or severe. The Chi-squared test with Yates' correction was used to compare the frequency of sore throat between the two groups.

Results

There were 20 patients in each group, with a comparable sex distribution of 11 men and 9 women in group A, and 12 men and 8 women in group B. The mean age of patients was 44.3 (SD 15.9) years in group A, and 42 (SD 15.5) years in group B. Table 1 lists the surgical procedures carried out. There is a comparable range of procedures between the two groups. Tracheal intubation was performed easily in all cases, and the duration of intubation averaged 53 minutes (SD 22.8) in group A and 54 minutes (SD 21.4) in group B.

Eighteen out of the 20 patients in group A developed a sore throat compared with only 10 out of 20 in group B (Table 2). This difference is statistically significant ($p < 0.01$). Of the 28 patients who experienced symptoms, only five classed their sore throat as moderate or severe; three of these were in group A and two in group B.

The influence of age, sex, duration of intubation and cuff pressure was also investigated (Table 3). There was no statistically significant association between the incidence of sore throat and any of these factors.

Table 2. Incidence of postoperative sore throat.

	None	Mild	Moderate	Severe	Total
Group A (hydrocortisone) (n=20)	2* (10%)	15 (75%)	1 (5%)	2 (10%)	18* (90%)
Group B (KY jelly) (n=20)	10* (50%)	8 (40%)	1 (5%)	1 (5%)	10* (50%)

*Chi-squared test significant at $p < 0.01$.

Table 3. Effect of age, sex, cuff pressure and duration of intubation on postoperative sore throat.

		Number	No sore throat	Sore throat
Cuff pressure	> 3.0 kPa	26	7	19 (73%)
	< 3.0 kPa	14	5	9 (64%)
Duration of intubation	> 59 minutes	17	3	14 (82%)
	< 59 minutes	23	9	14 (61%)
Age	> 40	18	5	13 (72%)
	< 40	22	7	15 (68%)
Sex	Male	19	4	15 (79%)
	Female	21	8	13 (62%)

Discussion

The idea of using a topical anti-inflammatory agent to prevent postoperative sore throat is not new. In 1958 Hamelberg studied the effect of applying lignocaine ointment that contained 1% hydrocortisone to red rubber tubes before their insertion.¹¹ His results, a decrease in the incidence of sore throat from 35% to 26%, were encouraging, but not statistically significant. Details of the anaesthetic technique and agents which were used in this study were unfortunately not given. However, there was no indication that these were standardised or that suxamethonium was avoided. Capan¹² has since demonstrated that sore throat is more frequent after the use of suxamethonium. He suggested that this may be a manifestation of suxamethonium myalgia. In addition, Hamelberg used an indirect questioning technique; he relied on the patients themselves volunteering symptoms related to the intubation. It is now appreciated that the incidence of sore throat varies with the method of questioning,^{3,13,14} and the preferred method in recent studies has been to ask the patients directly if they have experienced any sore throat.

The present study used this method, together with standardised anaesthesia and no suxamethonium, and has failed to confirm the encouraging results of Hamelberg. Indeed, the results show that the use of cream containing 1% hydrocortisone may actually increase the incidence of sore throat associated with tracheal intubation. The water-soluble hydrocortisone cream used contained chlorocresol, cetamacrogol emulsifying wax, liquid macrogol, white soft paraffin and liquid paraffin. None of these substances is particularly noted to be an irritant. However, it seems likely that one or more of them must have acted as such in this study.

The overall incidence of sore throat in the study (70%) is high, especially when compared to the Hamelberg study. There are three factors which may have contributed to this. Firstly, a direct questioning technique is known to produce a higher incidence than an indirect technique.¹⁴ Secondly, disposable PVC tubes were used in the present study, whereas red rubber tubes were used in the earlier study. The findings of other workers who compared tubes made from these two materials are inconclusive. Loeser,² Shah,¹⁵ and Sprague¹⁶ in their respective studies found that red rubber tubes were associated with a higher incidence of sore throat than PVC tubes, whereas Jensen⁵ found the reverse. It may be that the material used is unimportant. The differences found in the incidence of sore throat may merely reflect different designs (and hence area of contact between cuff and trachea) of the respective tubes.

Thirdly, the work of Loeser¹³ and Sprague¹⁷ has suggested that even bland lubricating jelly may increase the incidence of sore throat. Such a lubricant was used in the control group in the present study.

The finding that neither patient age, duration of intubation nor cuff pressure affect the incidence of sore throat supports the conclusions of previous studies.^{2,4} However, as

mucosal blood flow is compromised by lateral wall pressures exceeding 3 kPa,¹⁸ care should always be taken to limit cuff pressure to the 'just seal' level.

In conclusion, hydrocortisone cream joins a long list of both bland and active lubricants that have been shown to be ineffective in the prevention of postoperative sore throat. The author suggests that lubrication of tracheal tubes for this purpose is no longer appropriate.

Acknowledgments

The author thanks Dr J. M. Watt and Dr G. M. Cooper for their help with this study.

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Anaesthesia, 1990, Volume 45, pages 971-975

Isolated lung transplantation for pulmonary fibrosis

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Summary

The peri-operative anaesthetic management of 11 patients with pulmonary fibrosis undergoing single-lung transplantation is presented. Intra-operative problems, the early postoperative phase of recovery and intensive care, and other incidents in which general anaesthesia was required for the management of complications, are featured. Results, both short- and long-term, are mentioned. Major intra-operative events that cause concern appear to be related to the severity of the presenting illness and the development of respiratory failure. Others have reported the development of intra-operative cardiac failure. All cases were successfully managed operatively using conventional one-lung anaesthesia, although resort to partial cardiopulmonary bypass may have been indicated in some. The indications and attitudes to utilising cardiopulmonary bypass in the evolution of techniques for facilitating single-lung transplantation are reviewed.

Key words

*Anaesthesia; thoracic.
Surgery; lung transplantation.*

Lung transplantation has been attempted for more than 20 years. Success, judged by recipients consistently leaving hospital symptomatically improved, has been slow to achieve in comparison to other transplants. There is a gradual accumulation of world experience of single-lung transplantation for emphysematous and pulmonary vascular disease, but current evidence is that best results are achieved in patients with pulmonary fibrosis.¹⁻⁴ It is in these latter that both ventilation and perfusion favour the transplanted lung rather than the remaining, natural one.

The necessary surgery and anaesthesia are complicated by the poor state of health of recipients initially and by the major physiological changes that occur peri-operatively and that are brought about by surgical events and manoeuvres that facilitate the implantation of an orthotopic lung. We report data and events related to the peri-operative and postoperative management of 11 recipients of single-lung transplants for end-stage pulmonary fibrosis.

Methods

Patients. Details are shown in Table 1. There are six men and five women in the series; their ages at time of operation ranged from 27 to 58 (mean 45.8) years. Nine were diagnosed as suffering from idiopathic pulmonary fibrosis or cryptogenic fibrosing alveolitis. One patient had eosinophilic granuloma and another obliterative bronchiolitis after a bone marrow transplant 8 years earlier.

All patients were living a chair-bound existence before operation (mean Pao₂ breathing air 6.3 kPa) and required supplemental oxygen at rest. Minimal exertion resulted in oxygen desaturation, and prognosis for survival was estimated to be less than a year. One was dependent on positive pressure ventilation and had a tracheostomy. Examples of pre-operative data from the first six patients are shown in Table 2. Recipients had mild to moderate degrees of pulmonary hypertension, a degree of compromised right heart function, a restrictive ventilatory defect and marked impairment of diffusing capacity. Four were hypercarbic.

Surgery. All patients underwent left-lung transplantation. The pneumonectomy was followed by insertion of the transplant which was anastomosed in the sequence pulmonary veins (on a cuff of donor left atrium), pulmonary artery, and bronchus.⁵ The bronchus anastomosis was then protected with a wrap of omentum.

Anaesthesia. The anaesthetic technique was described previously.^{6,7} Six patients were sedated (midazolam, with or without droperidol) while peripheral venous, central venous (antecubital fossa), and arterial access were secured under local anaesthesia. Central neck lines were inserted after induction of anaesthesia with the patients in the head-down position. These patients are capable of generating large negative intrathoracic pressures and there is a danger of air embolus when they are breathing spontaneously.

Induction of anaesthesia was with a sleep dose of etomi-

Table 1. Patient details.

Number	Sex	Age (years)	Weight (kg)	Diagnosis	Transplant lung
1	F	43	49	Eosinophilic granuloma	Left
2A	M	54	68	Fibrosing alveolitis	Left
B			61	Rejection of first transplant	Right
3	M	58	65	Fibrosing alveolitis	Left
4	F	56	60	Fibrosing alveolitis	Left
5	M	54	82	Fibrosing alveolitis	Left
6	F	55	55	Fibrosing alveolitis	Left
7	M	27	45	Obliterative bronchiolitis	Left
8	F	46	52	Fibrosing alveolitis	Left
9	F	22	47	Fibrosing alveolitis	Left
10	M	44	73	Fibrosing alveolitis	Left
11	M	45	79	Fibrosing alveolitis	Left

date, followed by fentanyl and an intubating dose of vecuronium. A supplementary dose of alfentanil was given to ablate reflex responses to laryngoscopy immediately before tracheal intubation, and disposable right-sided double lumen tubes (Mallinkrodt, Bronchocath 37-39) were inserted to facilitate one-lung ventilation and anaesthesia.

Positive pressure ventilation with air and added oxygen was delivered by a Servo Elema ventilator (Model 900B). Inspired oxygen concentration was adjusted in accordance with cutaneous oxygen saturation values, and anaesthesia was maintained with enflurane or isoflurane. Vecuronium was used throughout the operation, and fentanyl was the intra-operative analgesic. All patients received a continuous infusion of dopamine (3–5 µg/kg/minute) during the peri-operative period.⁷

Pulse rate, ECG, cutaneous oxygen saturation, arterial blood pressure, oesophageal temperature and pulmonary artery pressure (from a flow-directed catheter sited in the main pulmonary artery)⁷ were monitored continuously. Later cases had a continuous monitoring fibre oxygen probe (Abbot Oxymetrix) inserted so that mixed venous oxygen could be measured continuously and cardiac outputs calculated by thermodilution. Arterial blood was sampled intermittently for assessment of blood gas status etc.

Postoperative management. Patients were transferred to

the intensive care unit for a period of elective ventilation. Muscle relaxants were stopped and patients sedated and kept free of pain with continuous infusions of opioids until they were haemodynamically stable, normothermic and ready to revert to spontaneous respiration. Suitability for cessation of ventilatory support was based ultimately on a trial of spontaneous ventilation through a Gilston T-piece when PEEP was no longer necessary. The patients had a peak ventilatory pressure of under 3.0 kPa and were maintaining normal arterial blood gas tensions with an inspired oxygen of 35% or less.

Results

Intra-operative. Table 3 summarises important data at set points when it was predicted⁷ there might be problems, namely, at induction of anaesthesia (Event 2); after institution of one-lung anaesthesia (Event 3); after clamping of the ipsilateral pulmonary artery and before recipient pneumonectomy (Event 4); and on perfusion and ventilation of the transplant (Event 5). Event 6 is the first results on return to the intensive care unit.

Induction of anaesthesia (Event 2) resulted in a slight decrease in systemic arterial pressure, but the institution of positive pressure ventilation may have contributed to the decrease since high inflation pressures were necessary to

Table 2. Examples of pre-operative data on first six patients.

Respiratory function											
Number	F _{IO} ₂	S _{PO} ₂	Blood gases			Forced expiratory volumes			Diffusion		
			pH	P _{CO} ₂ (kPa)	P _O ₂ (kPa)	FEV ₁ (litres)	VC (litres)	FEV/VC %	TL _{CO}	K _{CO}	
1	0.6	68				2.05	2.65	77	0.8	0.26	
2	0.3	87	7.41	5.7	7.1	2.1	2.35	89	1.63	0.53	
3	0.3	86	7.44	4.13	6.6	1.7	2.05	82	1.24	0.50	
4	0.2		7.40	6.1	7.9	0.8	0.9	88			
5	0.2		7.47	4.58	5.1	1.6	1.8	88	2.03	0.81	
6	0.6		7.40	6.7	16.3	0.75	0.9	83			
Cardiovascular function											
Number	Systemic pressure			Right atrial	Right ventricular	Pulmonary pressure			PCWP	TPR/PVR	CO
	Systolic	Diastolic	Mean			Systolic	Diastolic	Mean			
1	130	75	90	4/2	63/7	58	25	37	6	7.4/6.2	5.0
2	120	70	93	11/5	55/6	55	22	37	13	8.2/8.2	8.1
3	130	75	100	7/0	43/6	43	15	27	7	9.3/6.9	5.1
4	125	70	90	4/−4	45/4	45	15	27	7	7.7/5.7	5.7
5	110	75		8/1	60/10	60	30	42	8		5.2
6	110	70	85	4/0	42/4	42	18	29	8	6.6/4.8	

Table 3. Summary of intra-operative P_{aO_2} , P_{aCO_2} , and mean pulmonary artery pressure (MPAP) (SD).

Event*	P_{aO_2} (kPa)		P_{aCO_2} (kPa)		MPAP (mmHg)	
	Range	Mean	Range	Mean	Range	Mean
1	7.0–36.7	17.9 (8.9)	4.0– 8.5	6.2 (1.4)	—	—
2	7.6–61.4	23.7 (14.6)	4.3–14.4	8.4 (3.2)	21–57	35 (10.8)
3	5.6–31.3	14.2 (9.0)	4.4–14.6	7.8 (3.2)	21–51	36 (10.5)
4	5.3–32.2	14.4 (8.3)	4.5–13.2	7.9 (2.9)	23–63	44 (11.9)
5	7.8–35.6	14.5 (7.9)	4.0–10.8	6.5 (1.8)	22–42	31 (7.0)
6	9.6–22.4	14.8 (4.8)	5.1– 9.6	6.1 (1.5)	19–32	23 (4.3)

*Event: 1, patient sedated, breathing spontaneously; 2, two-lung ventilation; 3, one-lung ventilation, thoracotomy; 4, pulmonary artery clamped, recipient pneumonectomy; 5, two-lung ventilation, donor lung operational; 6, initial results on ICU.

ensure lung expansion. A moderate degree of head-down tilt restored normotension; fluid loads were not administered. Adequate oxygenation was achievable in all cases by increasing inspired oxygen in the ventilatory mix. Two patients required 100% oxygen to prevent desaturation to levels below 80% and most did so on institution of one-lung anaesthesia.

The commonest change in variables was an increase in the P_{aCO_2} . Changes above the accepted norm (> 5.5 kPa) occurred in six patients and levels continued to increase or remained elevated in five patients, despite increases in minute ventilation and attempts to find a more effective ventilation pattern.

The institution of one-lung anaesthesia (Event 3) was associated in two patients with significant decreases in oxygen saturation ($< 80\%$) that were not prevented by increased inspired oxygen. The saturation was less than 75% in one patient; resolution occurred when one-lung anaesthesia was stopped and the upper lung was temporarily ventilated with a jet ventilator attached to the left (tracheal) limb of the double-lumen tube. Arterial carbon dioxide tensions often remained elevated.

It was anticipated that pulmonary artery clamping (Event 4) would be the phase of operation which would prove critical. Pulmonary artery pressure values changed markedly in four patients, with peak values coming close to systemic values. The pulmonary artery vasodilator, usually sodium nitroprusside, exerted little effect independent of systemic pressure.

Pulmonary artery pressure decreased immediately in all but two patients when the transplanted lung was perfused, and in one previously reported patient,⁶ a precipitous decrease in oxygen saturation occurred. A large diversion of pulmonary blood into the transplant was manifest as a shunt in those with a high pulmonary vascular resistance in the natural lung. Oxygenation, as judged by a reduction in the inspired concentration necessary to maintain 80–90% saturation, improved slowly. Carbon dioxide levels returned rapidly to normal in those cases with hypercarbia.

There were five intra-operative events that were cause for concern. Two patients developed a tachycardia and non-specific arrhythmia with ST segment changes on perfusion of the transplant. Two developed severe intra-operative systemic hypotension. These were associated with pulmonary artery pressure levels similar to systemic values while receiving one-lung ventilation and while the transplant was being inserted. One patient developed a sudden bradycardia and nearly sustained a cardiac arrest.

The arrhythmic events were temporary and required no treatment. Circulatory disturbance was transitory. The association between transplant perfusion and arrhythmia production could be the result of several factors (e.g. the injection of cold perfusate from the transplant, air flushed into the left side of the heart from the transplant

pulmonary circulation at the time of blood flow restitution, or, most likely, electrolyte changes). The flush perfusate (Eurocollins) has a potassium concentration of 107 mmol/litre. In one patient a serum potassium of 6.5 mmol/litre was recorded within 5 minutes of the rhythm change. A left atrial sample immediately after transplant perfusion in another had a potassium concentration of 8.1 mmol/litre.

The severe hypotensive episodes occurred late in the operative period, after recipient pneumonectomy. Factors common to both events were hypercarbia ($P_{aCO_2} > 10$ kPa), respiratory acidosis ($pH < 7.15$) and raised pulmonary artery pressures (mean > 50 mmHg). Responses to a variety of drugs with potentially useful inotropic properties (calcium, dopamine, isoprenaline, aminophylline and adrenaline), were poor. The left femoral vessels were prepared for partial cardiopulmonary bypass to be instituted in both cases, but physiological stability began to return when the transplant was perfused and ventilated.

The sudden bradycardia developed immediately before the bronchus anastomosis was completed. This patient was hypercarbic, but despite a $P_{aCO_2} > 13$ kPa had been haemodynamically stable. The mean pulmonary artery pressure peaked at 50 mmHg, and the serum potassium was 5.0 mmol/litre when the cardiac rhythm changed. Internal cardiac massage for 3 minutes and adrenaline by intravenous infusion gave time for the bronchus anastomosis to be completed. Transplant ventilation and perfusion resulted in a rapid return to physiological normality.

After operation. All patients became cold during operation (mean at end 32.3°C). The decrease in body temperature was not prevented by the use of warmed gases, warm intravenous fluids, or a warming blanket. Factors that mitigate against preventing heat loss are the large amount of body surface exposed during surgery and the necessity for the liberal use of topical cold perfusate in the open hemithorax to minimise transplant warm ischaemia time.

Table 4. Postoperative results.

Number	Off ventilator at (time after operation)	Out of ITU at	Survival
1	12 hours	5 days	> 36 months
2A	44 hours	5 days	> 6 months
B	10 hours	5 days	7 weeks
3	5 days	13 days	> 32 months
4	28 hours	7 days	> 24 months
5	8 hours	3 days	> 22 months
6	47 hours	21 days	1 month
7	46 hours	5 days	9 months
8	15 hours	2 days	> 10 months
9	12 hours	36 hours	> 5 months
10	6 hours	36 hours	> 4 months
11	10 hours	36 hours	> 4 months

Ten patients were free from ventilatory support and their tracheas extubated within 48 hours of operation; one remained intubated for 5 days. The mean alveolar-arterial oxygen gradient at extubation was 15 (SEM 1.2) kPa. No patient was re-intubated except for further surgery. One patient sustained diathermy injury to the left phrenic nerve that delayed his weaning from ventilatory support. Another had a lower lobe infiltrate within a day of operation: it was considered that this was a residuum related to the donor. A third had to be returned to the operating theatre for omental wrapping of the bronchus. Patient No. 7 was weaned from ventilatory support at 2 days (tracheostomy de-cannulated at 3 days) despite artificial ventilatory dependence before operation. In no patient was re-ventilation necessary to cover episodes diagnosed as early rejection phenomena, although moderate oxygen desaturation (80%) was often concomitant.

Long-term problems. There are now eight survivors and three patients have died. The first of the latter did well initially despite a paralysed left hemidiaphragm, but his lung function began to deteriorate after 4 months. Open lung biopsy revealed chronic rejection obliterative bronchiolitis. This did not respond to conventional antirejection treatment. His right lung was transplanted 2 months later. One-lung anaesthesia (patient 2B) was conducted through a left-sided double-lumen tube, and utilised the first transplant lung for gas exchange. Immediate recovery was good and his trachea was extubated at 10 hours. However, re-transplantation did not halt a progressive rejection-initiated deterioration and the patient died of respiratory failure.

Two patients required repeat thoracotomies. A routine chest X ray on patient 5, taken on the 10th postoperative day, showed air in the pericardium. A diagnosis of bronchus anastomosis dehiscence was made. A right-sided double lumen tube was inserted after an etomidate, fentanyl and vecuronium induction. A separate jet ventilator was used to ventilate the left lumen of the tube and transplant lung once the left chest was open. Suitable operating conditions were achieved for repair of the bronchial anastomosis and the patient made an uneventful recovery.⁸

An isotope ventilation-perfusion scan of patient 6 showed that the natural lung remained preferentially perfused. Pulmonary angiography demonstrated an obstruction in the left pulmonary artery at the anastomosis; repair was necessary. Anaesthesia was conducted through a cuffed tracheal tube since one-lung anaesthesia was considered unnecessary. The pulmonary artery was clamped and a dacron graft was inserted to correct the vascular anastomosis. Recovery was uneventful, but after a few days it was dogged by problems of rejection. The patient died 3 weeks later. It is probable that the transplant sustained warm ischaemia at the time of re-exploration.

Discussion

This experience confirms earlier reports^{2,3,6} that conventional one-lung anaesthetic techniques are suitable for those patients suffering from pulmonary fibrosis who require a single-lung transplant. Alternative techniques for operation would require cardiopulmonary bypass.

Some of the intra-operative experiences reported, notably those associated with carbon dioxide retention, imply that operative survival would have been more certain had cardiopulmonary bypass been used. Reservations about invoking what is regarded as a safety net to the procedure have been based on concern for the consequences. The recipient pneumonectomy may be complicated because the primary pathology often results in the

formation of multiple adhesions. These may be compounded as a consequence of a previous therapeutic or diagnostic thoracotomy. It is believed that, under such circumstances, the introduction of anticoagulation to facilitate cardiopulmonary bypass would exacerbate bleeding problems.

A second concern relates to the virtual total dependence of the recipient on the transplant very shortly after implantation. Increase in lung water content is a concomitant of cardiopulmonary bypass. The transplanted lung is without an efficient mechanism to eliminate interstitial fluid since the lymphatics are sectioned at harvesting. We believe that the rapid return of these patients to spontaneous ventilation free from artificial support is related to rigid adherence to fluid restriction measures, even to the extent that renal function may be compromised. This must be maintained with diuretics and inotropes rather than fluid loads.

This judicious approach to the implementation of cardiopulmonary bypass is tempered by experience of two other single-lung transplants. Both patients were pathologically complex and use of cardiopulmonary bypass in their operative management was mandatory. Both died shortly after operation. One bled uncontrollably and the other developed intractable pulmonary oedema and died of complications.

DeMayo has addressed the problem of when cardiopulmonary bypass should be instituted.⁹ Bypass is recommended if the cardiac index decreases to less than 2.0 (litres/minute)/sq m; if the mixed venous oxygen saturation is less than 60%; if the mean pulmonary artery pressure increases above 45 mmHg when the pulmonary artery is clamped. The knowledge and implementation of such criteria, some of which were not measured in the early cases of our series because of doubts about the accuracy of the results,¹⁰ would have probably meant that most of the cases would have been prepared for bypass. Three would have had partial femorofemoral bypass introduced during operation. Early experience of the implementation of bypass on the basis of these criteria suggests that some of our reservations about its use in single-lung transplantation may be groundless (DeMayo, personal communication).

It is, however, our experience that some of the severe intra-operative problems are not just related to such cardiac function factors defined by the above data but are compounded by, and made critical in the presence of, the development of Type 2 respiratory failure. It is probable that neither cardiac failure nor the respiratory failure develop in isolation. It is our impression that it is those patients, in whom severe carbon dioxide retention is an early operative feature, who are likely to be particularly problematical during transplantation operations.

The long-term results in these cases are yet to be fully evaluated. World experience of single-lung transplantation for pulmonary fibrosis is now in excess of 50 patients and a survivor of 5 years' duration was reported.¹¹ The sum of such experience, and that reported in this analysis, continue to support the original premise and the previous experience. This suggests that selected cases suffering from end-stage pulmonary fibrosis, with reasonable right ventricular function can withstand an orthotopic, isolated lung transplant operation conducted with conventional one-lung anaesthesia without a need to resort to the use of cardiopulmonary bypass.

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Anaesthesia, 1990, Volume 45, pages 975-978

pH-adjustment and discomfort caused by the intradermal injection of lignocaine

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Summary

One hundred adult day-case patients who required intravenous access had cannulae inserted using local anaesthesia with 1% lignocaine, 1% lignocaine with adrenaline or the corresponding pH-adjusted solutions. The local anaesthetic solutions were modified by the addition of 1 ml 8.4% sodium bicarbonate to 10 ml lignocaine. Pain scores at different stages of cannulation were noted and showed a significant reduction after use of pH-adjusted solutions ($p < 0.02$ for the plain lignocaine, and < 0.001 for the lignocaine with adrenaline). Modification of the pH of lignocaine solutions by the addition of sodium bicarbonate is a simple method significantly to reduce the discomfort caused by the infiltration of the local anaesthetic.

Key words:

Anaesthetics, local; lignocaine.
Complications; pain.

Local anaesthetics, although used for analgesia, can themselves cause considerable discomfort especially on skin infiltration.¹⁻³ Local anaesthetic solutions are more soluble and stable at an acidic pH. However, raising their pH by the addition of sodium bicarbonate results in enhanced efficacy when the anaesthetic is used for regional blockade.^{4,6} It has also been noted empirically that lignocaine that has been pH-adjusted by addition of sodium bicarbonate does not 'sting' on skin infiltration.⁷

This double-blind prospective study was designed to assess the effect of the addition of sodium bicarbonate to lignocaine solutions on pain caused by skin infiltration.

Methods

Approval for the study was obtained from the Trafford Ethics Committee. One hundred unpremedicated adult patients who presented for day-case procedures that would require intravenous access were studied. Verbal consent was obtained on the day ward.

The modified local anaesthetic solutions were prepared by adding sodium bicarbonate to 1% lignocaine and 1% lignocaine with 1:200 000 adrenaline in the ratio of 1 ml of 8.4% bicarbonate to 10 ml of the local anaesthetic. The bicarbonate was obtained from a 10-ml paediatric minijet, and added in the same proportion as that used for regional block at the University of Virginia Medical Center.^{4,7} The pH and osmolality of these solutions were measured before the start of the study (Table 1).

The patient was then randomly allocated (by means of a random number generator) in a double-blind manner to receive one of the four prepared local anaesthetic solutions. The skin on the dorsal aspect of the patient's nondominant wrist was cleaned with 70% isopropyl alcohol and allowed to dry in preparation to cannulate a vein. A 1-ml insulin syringe with a microfine III needle (0.36 mm by 12.7 mm), that contained one of the local anaesthetic solutions was introduced intradermally. The patient was asked to indicate any discomfort verbally on being shown a descriptive fixed-interval four-point pain scale, after the needle had been

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This paper was awarded the President's Medal after being presented for the Registrars' Prize at the 1989 Annual Scientific Meeting of the Junior Anaesthetists' Group of the Association of Anaesthetists.

Accepted 11 April 1990.

Table 1. Group allocation and pH and osmolality of the local anaesthetic solutions.

Group	pH*	Osmolality* (osmol/kg)
1% plain lignocaine (Antigen)	4.95	70
1% lignocaine with adrenaline 1 : 200 000 (Astra)	3.76	273
1% lignocaine and sodium bicarbonate†	7.54	230
1% lignocaine with adrenaline and sodium bicarbonate†	7.39	406

*pH was measured using a Corning 140 digital pH meter, osmolality using an Advanced Instruments digimatic 3D2 osmometer.

†Sodium bicarbonate was added in the proportion of 1 ml 8.4% bicarbonate to 10 ml local anaesthetic solution.

introduced into the skin. The choices were none, mild, moderate, and severe and were scored 0 to 3 accordingly.

The skin was then infiltrated with 0.1 ml of the local anaesthetic solution and again the patient was asked to indicate any discomfort from the scale. Ten seconds were allowed to pass and the vein cannulated with an 18-gauge cannula. Discomfort was again assessed, first on puncture of the vein, and then finally as the cannula was threaded up the vein. The patient's results were excluded if the cannula did not enter the vein at the first attempt.

The pain scores for each step of cannulation were examined between groups by the Kruskal-Wallis analysis of variance. The Mann-Whitney *U* test was used to compare infiltration scores for plain versus adjusted lignocaine and lignocaine with adrenaline versus adjusted lignocaine with adrenaline. A probability value of < 0.05 was taken to be statistically significant.

Results

The groups were comparable in terms of age, weight, and sex distribution. There was an overall preponderance of males to females in the study in the ratio of 3/1 (Table 2).

There was no significant difference between groups in the pain scores for needle insertion, vein puncture, and cannula advancement (Table 3). In contrast, analysis of variance for infiltration scores indicated a highly significant ($p < 0.001$) difference between groups (Table 4). The addition of bicarbonate reduces pain on infiltration, and with the Mann-Whitney *U* test the difference in pain scores between unmodified and pH-adjusted solutions were shown to be statistically significant (Table 5).

Discussion

The excess of male patients in the study was because 69% of patients overall presented for orthopaedic surgery, and

these were largely young adult males for arthroscopy (Table 2).

A four-point fixed-interval pain scale was used since this was considered to be the most practical way to assess pain quickly at each of the stages of venous cannulation. The major criticism of a four-point pain scale is its lack of sensitivity,⁸ but this does not seem to have been detrimental in terms of significant differences found. It is stated, in a survey on the measurement of pain, that the four-point scale 'remains a useful standard method with the advantage of simplicity'.⁹

The results of this study show that in unpremedicated patients, the addition of sodium bicarbonate to plain lignocaine and to lignocaine with adrenaline reduces discomfort caused by the local anaesthetic on infiltration. This confirms the results of two volunteer studies recently published in the United States.^{10,11} The pain scores and their distribution for most of the stages of cannulation were very similar; $> 90\%$ patients experienced none or only mild discomfort. However, 36 to 46% of patients experienced moderate or severe discomfort on infiltration with the unmodified local anaesthetic. This does not mean that local anaesthetic should not be used, since it was shown that even without pH adjustment, lignocaine infiltration reduces the pain of percutaneous venous cannulation.¹²⁻¹⁴

It is understandable that needle insertion into the skin, infiltration, and puncture of the vein wall should cause some discomfort, but it is harder to see how cannula advancement along the vein should be uncomfortable; it is probably associated with irritation or even stripping of the intima.

The mechanism of the decrease in pain on infiltration with bicarbonated lignocaine is unclear at present. It seems likely that it is related to an increase in pH. There are inconsistencies with this being directly related to acidity, however, since procaine and chlorprocaine are more acidic but less painful on skin infiltration than lignocaine.^{2,3}

It may be that since the pH change increases the uncharged basic form of the drug, it increases its diffusion through tissues. This will result in a higher concentration of the drug in the nerve axoplasm and a more rapid block of the sensory fibres, as suggested by Christoph *et al.*¹¹ However, examination of the pKas of various local anaesthetics does not seem to be related to painful infiltration.³ The same paper noted that lipid solubility most closely paralleled painful infiltration but that the relationship was not precise. Thus the exact mechanism is not yet obvious.

Alkalinisation reduces the solubility of local anaesthetics and made precipitation after pH adjustment with bicarbonate a possibility. Five millilitres of 8.4% bicarbonate were added to 5 ml 1% lignocaine and 1% lignocaine with adrenaline before the start of the study, that is in a proportion 10 times greater than used in this study, with no evidence of precipitation. This does not apply to all local anaesthetics, for example, only 0.25 ml 8.4% bicarbonate added to 10 ml 0.5% bupivacaine with 1 in 200 000 adrenaline will result in a permanent precipitate.⁶ pH-adjusted

Table 2. Patient data.

	Number of patients in group	Sex		Mean age in years (SD)	Mean weight in kg (SD)
		Male	Female		
1% plain lignocaine	22	19	3	38 (17)	73.7 (11.5)
1% lignocaine with 1 : 200 000 adrenaline	28	19	9	43 (20)	71.8 (10.4)
1% lignocaine and sodium bicarbonate	26	19	7	39 (17)	69.6 (13.8)
1% lignocaine with adrenaline and sodium bicarbonate	24	18	6	36 (19)	69.9 (10.2)
Totals	100	75	25	39 (18)	71.2 (11.5)

Table 3. Pain scores for needle insertion, vein puncture, and cannula advancement.

	Pain score (number of patients)				Total	
	0	1	2	3		
Needle insertion						
1% plain lignocaine	8	12	2	0	22	H=4.75 (ns)*
1% lignocaine with 1 : 200 000 adrenaline	14	13	1	0	28	
1% lignocaine with sodium bicarbonate	17	9	0	0	26	
1% lignocaine with adrenaline and sodium bicarbonate	12	9	3	0	24	
Totals	51	43	6	0	100	
	Pain score (number of patients)				Total	
	0	1	2	3		
Vein puncture						
1% plain lignocaine	17	5	0	0	22	H=0.56 (ns)*
1% lignocaine with 1 : 200 000 adrenaline	20	5	2	1	28	
1% lignocaine with sodium bicarbonate	20	5	1	0	26	
1% lignocaine with adrenaline and sodium bicarbonate	18	3	2	1	24	
Totals	75	18	5	2	100	
	Pain score (number of patients)				Total	
	0	1	2	3		
Cannula advancement						
1% plain lignocaine	11	7	3	1	22	H=3.62 (ns)*
1% lignocaine with 1 : 200 000 adrenaline	20	8	0	0	28	
1% lignocaine with sodium bicarbonate	16	7	3	0	26	
1% lignocaine with adrenaline and sodium bicarbonate	14	9	1	0	24	
Totals	61	31	7	1	100	

*Analysis of variance between groups for each separate step of cannulation.

Table 4. Infiltration pain scores.

	Pain score (number of patients)				Total	
	0	1	2	3		
Infiltration						
1% plain lignocaine	5	9	8	0	22	H=29.31* (p<0.001)
1% lignocaine with 1 : 200 000 adrenaline	1	14	10	3	28	
1% lignocaine with sodium bicarbonate	13	11	2	0	26	
1% lignocaine with adrenaline and sodium bicarbonate	16	6	2	0	24	
Totals	35	40	21	4	100	

*Analysis of variance indicating a highly significant difference in pain scores between groups for local anaesthetic infiltration.

Table 5. Between-group differences in infiltration pain scores and comparisons of modified and unmodified solutions.

Mean	Pain score		Comparison using Mann-Whitney <i>U</i> test
	Median		
1% plain lignocaine	1.1	1	1% lignocaine as compared with 1% lignocaine with sodium bicarbonate (p<0.02)
1% lignocaine with sodium bicarbonate	0.6	0.5	
1% lignocaine with 1 : 200 000 adrenaline	1.5	1	1% lignocaine with 1 : 200 000 adrenaline compared with 1% lignocaine with adrenaline and sodium bicarbonate (p<0.001)
1% lignocaine with adrenaline and sodium bicarbonate	0.5	0	

local anaesthetic solutions are less stable than unmodified solutions, although when the amount of bicarbonate added is limited and the pH is kept close to 7, alkalised bupivacaine and adrenaline were shown to be stable for up to 6 hours.¹⁵

The implications of this study are wider than just in the context of venous cannulation and may be useful for infiltration for minor surgery, and in the use of local anaesthesia in the Accident and Emergency department. Modified local anaesthetic solutions may also have a place in paediatric practice in situations where there is not sufficient time to use EMLA cream.

Acknowledgments

I thank the nursing staff on the orthopaedic day ward of Trafford General Hospital, J. Jeacock, and L. Hunt, statisticians with the University of Manchester, for their help and advice with the statistics and Dr J. Kinsella, Consultant Anaesthetist at Trafford General Hospital for his help with the method of randomisation in the study.

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Anaesthesia, 1990, Volume 45, pages 978-980

Was CEPD right?

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Summary

This retrospective study found that the long-term (greater than 6 months) postoperative survival in ASA 4E and 5E patients was 41% and 21% respectively, in 1986. This supports the Confidential Enquiry into Peri-operative Deaths' recommendation that life-saving surgery should not be withheld from patients who present in so serious a condition that they are unlikely to survive surgery.

Key words:

Complications; death.

The report on a Confidential Enquiry into Peri-operative Deaths¹ (CEPD) published in 1987 made several recommendations with which anaesthetists and surgeons are now familiar. Among them was the recommendation that a patient who presents in so serious a condition that survival is unlikely whether or not surgery is undertaken should not be operated upon, preserving dignity in death and avoiding

unnecessary suffering.² The decision not to operate should be taken jointly by consultants or senior registrars in anaesthesia and surgery. These conclusions were reached because the pre-operative prognosis was poor in a large number of patients who died early in the postoperative period. However, cases were considered only if the patient died. The assessors were unable to consider patients whose

Table 1. Age and gender of patients in ASA groups 4E and 5E.

Age (years)	4E		5E	
	M	F	M	F
65-69	2	—	1	—
70-74	1	—	5	4
75-79	3	1	7	2
80-84	2	3	3	2
85-89	—	2	1	2
90-94	—	1	—	1
Total	8	7	17	11

pre-operative prognosis was equally poor but who survived; that is, they lacked denominator data. This retrospective review was undertaken in an attempt to investigate this recommendation of the CEPOD report.

Method

Patients aged 65 years or more who had undergone emergency major surgery at Queen's Medical Centre, Nottingham, were identified from the 1986 operating theatre registers in an attempt to find those most likely to be classified as ASA 4E or 5E during the same period as the CEPOD study. The relevant notes were retrieved and examined when such a case was identified. Our hospital's anaesthetic record includes a section for pre-operative assessment, including an ASA scale. This was studied, together with the notes pertaining to that admission. The case was included in the study only if it was confirmed that the patient fulfilled the criteria for consideration as ASA 4E or 5E.

The following data were recorded: age, sex, and ASA status; pre- and postoperative diagnoses; the features that caused the patient to be considered as ASA 4E or 5E; postoperative survival time.

Results

Altogether 509 patients were identified from the operating theatre registers. Case notes for 402 (79%) of these were found. Forty-three could be classified unequivocally as being ASA 4E or 5E at the time of surgery. Table 1 shows these 43 cases classified by ASA grade, age and gender.

Pre- and postoperative diagnoses. Generally there was agreement between the anticipated diagnosis and the findings at surgery. An additional diagnosis of carcinoma of the colon was made in one patient during a laparotomy for overseeing of a perforated duodenal ulcer, and in one case

Table 2. Pre-operative surgical diagnoses.

Haemorrhagic causes (all systems)	Leaking aortic aneurysm	7
	Bleeding peptic ulcer	4
	Intracranial haemorrhage	3
	Haemorrhagic pancreatitis	1
	Bleeding prostatic bed	1
	Bleeding bladder wall	1
	Bleeding fibroid	1
Gastrointestinal	Perforated duodenal ulcer	7
	Other perforated bowel	3
	Mesenteric embolus	3
	Intestinal obstruction	2
	Strangulated hernia	2
Other vascular	Femoral embolus	2
	Brachial embolus	1
Skeletal	Fractured femoral neck	5

Table 3. Frequency of complicating conditions.

Cardiovascular	Hypovolaemia	22
	Arrhythmia	11
	Cardiac failure	9
	Ischaemic heart disease	8
	Peripheral vascular disease	5
	Coagulopathy	3
	Digoxin toxicity	3
	Recent myocardial infarction	2
	Hypertension	2
	Chronic obstructive airways disease	11
Respiratory	Pneumonia	10
	Gastric aspiration	2
Renal	Pulmonary embolus	1
	Acute failure	6
Metabolic	Chronic failure	3
	Carcinomatosis	3
	Diabetes mellitus	2
	Hypokalaemia	2
	Hyperkalaemia	1
Generalised sepsis		2
Morbid obesity		1

of intestinal obstruction there were no abnormal findings at laparotomy. Otherwise the pre- and postoperative diagnoses were the same. Table 2 lists the pre-operative diagnoses.

Associated conditions. Table 3 lists the additional conditions that resulted in the classification of ASA 4E or 5E. Most patients presented with more than one of these. It was not possible to identify meaningful postoperative survival times from four of the case notes. Some patients were returned to the referring hospital, or discharged to a long stay institution, within a few weeks of surgery. Patients were discharged home in other cases, but failed to attend outpatient clinics and were lost to follow-up. It is possible that some may have died at home. Data from these patients were disregarded. One ASA 4E patient was discharged from follow-up care 2 months after surgery, and two ASA 5E patients were discharged at 1 and 3 months respectively. These three patients are also disregarded. Four patients died during surgery. Table 4 shows the survival times and ASA groups of those patients for whom complete data were available.

One each of the ASA 4E and 5E patients who survived for more than 6 months was still alive at the time of writing (September 1989).

Discussion

This study was prone to all the pitfalls of retrospective analysis of data that had not been collected in a structured manner. Some of these difficulties were overcome by disregarding patients for whom the available data were incomplete. It was not possible to collect any true denominator data; it is not known how many ASA 4E and 5E patients

Table 4. Duration of survival after surgery. Four ASA 5E patients died during surgery.

Survival	ASA	
	4E	5E
0-9 days	3	16
10-19 days	2	—
20-29 days	2	—
1-2 months	1	1
2-6 months	1	1
> 6 months	4	5
Total	13	23

were admitted to surgical beds but did not undergo surgery. In addition, only 402 of the 509 case records requested could be found. However, from the data available to us, we found that 43% of patients classified as ASA 4E or 5E who underwent surgery survived for 30 days or more. By concentrating our study on these groups of patients we have included those patients classified in CEPOD as 'moribund'.

The CEPOD report noted that there were important differences in clinical practice between the centres involved in their study.⁴ The patients examined in this study underwent surgery during 1986, to coincide with the period of the CEPOD study.⁵ Despite the differences between centres, the state of knowledge was the same when these and the CEPOD patients were being treated, and the CEPOD recommendations could not have influenced the treatment given to any patient in this study.

The majority of the patients considered in the CEPOD study were aged 65 years or more.⁶ A similar age group was chosen for this investigation. No attempt was made to assess retrospectively the various factors that may have influenced outcome nor to scrutinise the practice of each specialty.

Table 4 shows that the death rate was higher in the ASA 5E than 4E group and that most deaths occurred soon after surgery, as has been observed previously.^{7,8} The majority of patients who survived for longer than 30 days were still alive 6 months after surgery. Our numbers are very small when compared to those studies, and the survival time bands we have chosen are not always directly comparable. One study⁷ found that the 48-hour postoperative mortality in ASA 4E and 5E patients was 8.3% and 9.5% respectively; those patients were not classified according to age. That study also provided figures for ASA 5 patients who had elective surgery. Another study⁸ found the one-week postoperative mortality in ASA 4 and 5 patients was 23%

and 51% respectively; 63% of ASA 4 and 75% of ASA 5 patients had emergency procedures.

The 10-day mortality in ASA 4E and 5E patients in the present study was 23% and 70% respectively. Our figures show a 41% survival beyond 6 months in ASA 4E patients, and a 22% survival beyond 6 months in ASA 5E patients.

The original recommendation was restated in a circular issued by the CEPOD Office,⁹ and suggested that only nonlife-saving surgery should be withheld. The results of our study suggest that life-saving surgery is justified in ASA 4E and 5E patients.

Acknowledgments

We thank Mr B. Feurtado, Medical Records Research Assistant at the Queen's Medical Centre, Nottingham, without whose help this study would not have been possible.

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Two locum anaesthetists convicted of manslaughter

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On 26 January 1990 Dr John Adomako was convicted of manslaughter at the Old Bailey after the death of a patient at the Mayday Hospital from cerebral hypoxia in July 1987. Adomako was, however, acquitted of two counts of perjury relating to evidence given by him in the Coroner's Court where *inter alia* he denied leaving the patient unattended in the operating theatre while he went to get himself a coffee and switching off the alarms on the ventilator. His notes were inadequate and full of gaps.

Adomako was born in Ghana on 28 August 1940 but trained in Russia, qualifying at Rostov on Don in 1972. On arrival in Britain and for some years afterwards, his basic medical knowledge was considered deficient by some senior doctors who employed him. Notwithstanding and despite the fact that he never acquired any postgraduate qualifications in anaesthesia, he had worked in Britain since about 1972 in a variety of, mainly locum posts in anaesthesia. In 1975, when an SHO, he had been reprimanded for failing to attend lectures. He had never joined the Association of Anaesthetists of Great Britain and Ireland. The Mayday Hospital had not taken up references for his locum appointments as an anaesthetic registrar since the posts were considered of too short duration to warrant this.

At the coroner's inquest which was adjourned so that criminal prosecution could be brought, Adomako was confronted with evidence and the allegation that he had left the patient anaesthetised during the operation to repair a detached retina while he went to have a cup of coffee. He was accused of gross negligence and disregard for the patient. He was convicted of manslaughter and sentenced by Mr Justice Allott to 6 months in prison, suspended for one year. His limited registration was suspended when he was charged and is not likely to be renewed by the General Medical Council.

The background facts

On Sunday 4 January 1987, Alan Loveland, a 33-year-old, healthy, married man was due to undergo surgery to repair a detached retina at the Mayday Hospital in Croydon. Dr Ahmed Hassan Said, the anaesthetic registrar induced anaesthesia at 0945 hours using fentanyl 100 µg and thiopentone 400 mg. Vecuronium 8 mg was administered to produce muscular paralysis, after which a No. 9 Mallinckrodt tracheal tube was passed to facilitate control of the patient's breathing through connexion to a Pulmovent anaesthetic lung ventilator. His condition was monitored by the use of a Simonsen and Weel electrocardioscope (ECG); a Critikon 'Dinamap' model 1846; a peripheral nerve stimulator to assess the degree of paralysis produced by the vecuronium. He also used an anemometer

(a Wright's Respirometer) in the expiratory breathing hose to measure the volume of expiration. The patient breathing system was also connected to a ventilator failure alarm.

The operation proceeded routinely at first, but at about 1030 hours Dr Said was required to attend at an emergency Caesarean section along with his operating department assistant (ODA). He did not call an anaesthetist from home, but summoned Dr Adomako, the locum on duty, to take over from him. An ODA (to replace the ODA who had to leave the theatre with Dr Said), arrived approximately 20 minutes after the changeover occurred. The changeover took place at a time when the lighting in the theatre was dimmed and the two operating surgeons, a consultant and registrar were not informed of, and did not know about, the changeover of anaesthetists until the patient's condition had so far deteriorated as to give cause for serious concern. (The theatre lighting had been turned up from its previously dimmed levels necessary for the operation for about 20 minutes before the patient was discovered to have suffered a cardiac arrest and all the hospital's equipment was found to be in good working order).

At about 1035 hours Dr Said gave an increment of vecuronium 2 mg in the presence of Adomako when he was handing over the care of the patient to him. Dr Said demonstrated the need for more of this drug using the neuromuscular stimulator. He left the ventilator failure alarm switched on; a mute switch could suppress the alarm for about 20 seconds whereafter it would automatically sound again. The audible alarm could only be permanently suppressed by switching off the alarm unit using the on/off key.

Ten minutes or so after taking over from Dr Said, the anaesthetic record showed that Adomako gave another 4 mg of vecuronium to intensify the paralysis because he thought the patient was a bit 'light'.

Professor A. P. Adams, who gave evidence at the trial, considered that this 'lightness' may well have been the patient reacting to hypoxia occasioned by disconnection of the breathing system which had gone unnoticed (no alarm had sounded). At 1110 hours the record showed a pulse rate of 40/minute and blood pressure 70 mmHg and that Adomako administered atropine 0.3 mg at 1115 hours followed by a second dose of the same amount. Adomako did not attempt to check any of the ventilator hose connexions at the patient's end of the system, although all he had to do was to run his hand up alongside the hoses under the sterile towels towards the head end of the operating table, which he should have done. Yet, 'Disconnection of a part of the breathing system is the most frequent of the various problems encountered during an anaesthetic and

thus should be one of the first things to consider in a crisis.' Also, in any crisis like this, the anaesthetist should ventilate the patient's lungs by hand to ascertain the feel of the patient's lungs and to expose any problems.¹

Adomako's evidence of what had occurred during the critical period was not in keeping with the evidence given by the ODA who said that when he entered the operating room at 1045 or 1050 hours the blood pressure alarm was sounding. Adomako, the ODA said, thought the machine was not working properly, but having obtained another blood pressure cuff, no BP reading was obtainable. At this point the ODA went to fetch atropine and was told by a nurse that Adomako wanted to set up an intravenous drip. Adomako was unable to establish an infusion in the left forearm but ultimately inserted a cannula into a vein on the right forearm. While these attempts were being made, the ODA noticed that the patient's hands and fingernails were slightly blue. Simultaneously, the surgical registrar, who had noticed the heart rate to have fallen to below 30 beats/minute, removed the drapes covering the patient and found him 'absolutely blue'. There was no carotid pulse so he began cardiac massage; as he did so he noticed the ventilator was not connected to the tracheal tube which he pointed out to Adomako who was still trying to establish the intravenous infusion on the right forearm. The ventilator was reconnected and the patient's lungs ventilated manually with oxygen.

The patient was thereafter 'successfully' resuscitated but was later diagnosed as having suffered severe and irreversible brain damage, from which he died some 6 months later without ever recovering consciousness. This was consistent with a disconnection from the ventilator for several minutes; further, the signs of such event were evident during this period (decreased BP and pulse rate) which culminated in the audible warning alarm from the Dinamap, and finally asystole, visible on the ECG. Adomako made no mention of the patient's cyanosis in his notes and only admitted to it under cross examination. Adomako had called in the cardiac arrest team, yet by the nature of his training and experience, he should have been able to handle the crisis better than a group of junior doctors.

Although Dr Adomako was employed for a number of short-term posts as a locum registrar anaesthetist, few if any references were taken up by his employers who were

willing to engage him as a locum registrar in a specialty for which he possessed no qualifications. The taking-up of references was apparently considered to be the responsibility of the locum agency concerned and one manager stated that formal references were not obtained 'as he came to this agency from a permanent post at Crawley Hospital'.

It was the trial judge's view that the responsibility for the patient's death lay wider than simply with Dr Adomako's personal incompetence and neglect. The Mayday Hospital's operating theatre was well equipped but its call arrangements were less satisfactory, and these have now been modified. It is to be hoped that no locum posts will be filled without references being taken up by the employing authority, and that the doctors taken on will be adequately trained for the job, and not moonlighting from another stressful and over-full-time position.

Since Dr Adomako was convicted in London, a second anaesthetist, 66-year-old Dr Norman Sargent has also been convicted of manslaughter following the death of a 55-year-old woman in June 1988. Dr Sargent was acting as a locum consultant when he connected the patient to a high pressure oxygen source, namely, a cylinder containing oxygen at 2000 lb per square inch (psi); the cylinder was three-quarters full. That pressure was reduced to 15–20 psi. The prosecution claimed that the entire contents of the cylinder were emptied into the patient via a tracheal tube causing her to become inflated until she resembled the 'Michelin Man' advertisements and she died from barotrauma. Dr Sargent did not give evidence. He was found guilty of manslaughter and, like Adomako, sentenced to 6 months imprisonment suspended for one year. Dr Sargent, although employed as a locum consultant, held only basic qualifications, namely he was a licentiate of the Worshipful Society of Apothecaries and had a diploma in anaesthetics.

The conviction of Dr Adomako and Dr Sargent of manslaughter for gross neglect or dereliction of their duty to the patient marks a new stage in the accountability of doctors. Arguably, Dr Adomako's basic lack of competence and specialist training should have ensured that he was not given sole and unsupervised responsibility for patients' lives.

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Phonation affects Mallampati class

We were interested to read the letter by Drs Wilson and John (*Anaesthesia* 1990; 45: 486-7) which suggests that phonation may be a cause of error when the Mallampati classification is performed. We examined a subgroup of 366 patients scheduled for elective surgery in an unpublished prospective study. Each patient was scored for Mallampati class.¹ This was repeated with the patient saying 'Ah'. Laryngoscopy was performed on 334 of these patients. The view at laryngoscopy was graded from 1 where all of the laryngeal structures were visible to 5 where not even the epiglottis was seen, as described by Wilson and colleagues.² Grades 4 and 5, that is where no more than the epiglottis was visible, were defined as difficult laryngoscopy.

Thirty-six of the 74 patients who were scored Mallampati class 3 without phonation, became class 1 when saying 'Ah'. The view of the pharyngeal structures worsened from class 1 to class 2 in two patients. Six out of 334 patients (1.8%) were difficult at laryngoscopy, one of whom was predicted to be difficult using the Mallampati classification (Table 1). When this was performed with the patients saying 'Ah' none of the six difficult laryngoscopies was predicted (Table 2).

The tongue is flattened and the paired *levator veli palatini* muscles contract during phonation, and the soft palate is pulled upwards and backwards. The position of the soft palate is higher during the production of closed vowels (ee, oo) than open vowels (ae, ah).³ This improves the view of the pharyngeal structures on which the Mallampati classification is based.

We, in common with Wilson and John, have found

Table 1. Mallampati class (1 to 3) and laryngoscopy grades (1 to 5) in 334 patients.

		Laryngeal view				
		1	2	3	4	5
Mallampati Class	1	187	37	5	4	0
	2	32	2	0	1	0
	3	43	22	0	1	0

Table 2. Mallampati class (1 to 3) and laryngoscopy grades (1 to 5) in 334 patients. Mallampati classifications was performed with the patient saying 'Ah'.

		Laryngeal view				
		1	2	3	4	5
Mallampati 'Ah' Class	1	235	51	4	6	0
	2	11	3	1	0	0
	3	16	7	0	0	0

substantial interobserver variation in assessing Mallampati class (unpublished observations). Our findings suggest that if a patient inadvertently phonates while being assessed, the Mallampati class may be altered. We considered whether the Mallampati 'Ah' test could be used to screen out false positives and reduce interobserver variation in the Mallampati classification. Our results indicate that while this might be the case, it could be at the expense of an increase in false negatives. Confirmation of this would require a large prospective study given the low incidence of difficult laryngoscopy and high false negative rate we have found with the Mallampati test.

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Regional anaesthesia must be properly managed

The May 1990 issue of this Journal contained a number of items that concern me as a proponent of regional anaesthesia. These techniques have much to offer our patients, but they must be used properly. Their use implies a lot more than just needle and catheter insertion and drug injection because the patient must be managed correctly, not only during the operative procedure, but before and after it as well. This implies a continuous process of decision-making with sensitive handling of the patient and the avoidance of complications. Against such a background some of the issues raised can be considered.

Professor Aitkenhead in his Editorial on awareness (*Anaesthesia* 1990; 45: 351-2) indicates that patients have complained of either pain or an 'unnecessary' general anaesthetic when a block was not perceived as totally successful. Such problems stem from a number of causes, notably total reliance on somatic nerve block to cover all the discomforts that may arise during surgery. A good block is essential, but apart from a few particular situations, the planned administration of sufficient sedative or anaesthetic drug to ensure unconsciousness has everything to commend it and avoids such difficulties.

Careful management is needed when the patient is to be conscious, (especially during Caesarean section), but I am concerned by his bald advocacy of a routine warning of pain or discomfort. Patients must realise that they will know what is happening, and that some obstetric manoeuvres, particularly, can cause discomfort. The patient is advised to tell an anaesthetist of any concern as the block develops and reassured that this will be dealt with (by general anaesthesia if ultimately necessary). Anxiety is often the root cause of the problem in such situations, and is there really any objection to the use of small doses of systemic sedative or analgesic drugs to deal with many problems? No patient should suffer distress during surgery no matter what type of anaesthetic is involved.

Drs Wulf and Striepling (*Anaesthesia* 1990; 45: 357-61) describe histological changes in the epidural spaces of patients who received postoperative analgesia by that route. They issue severe warnings about a number of contraindications to extradural block while admitting that the changes they observed 'have little clinical significance'. In spite of that I would admit to some concern about the use of this form of analgesia in many of their patients,

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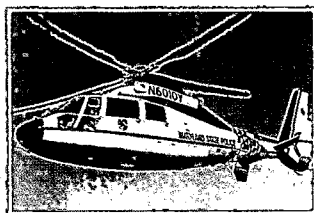
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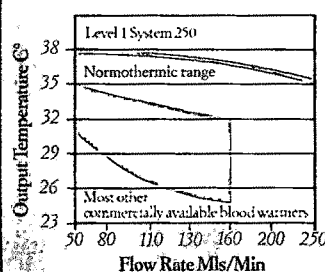
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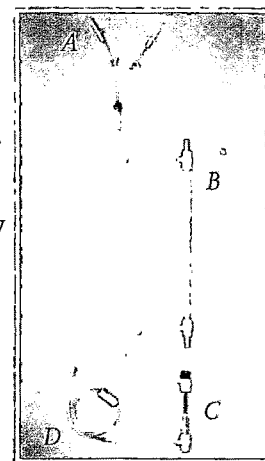
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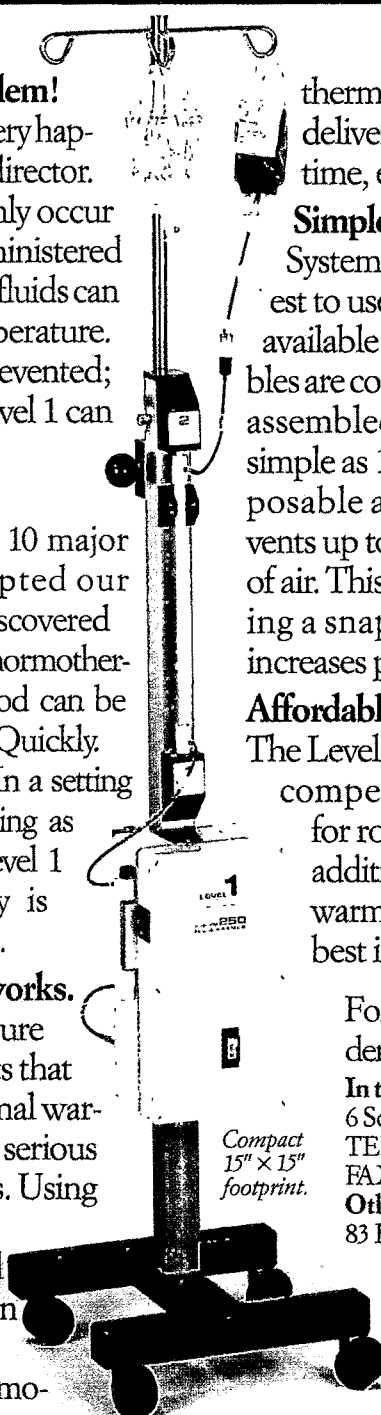
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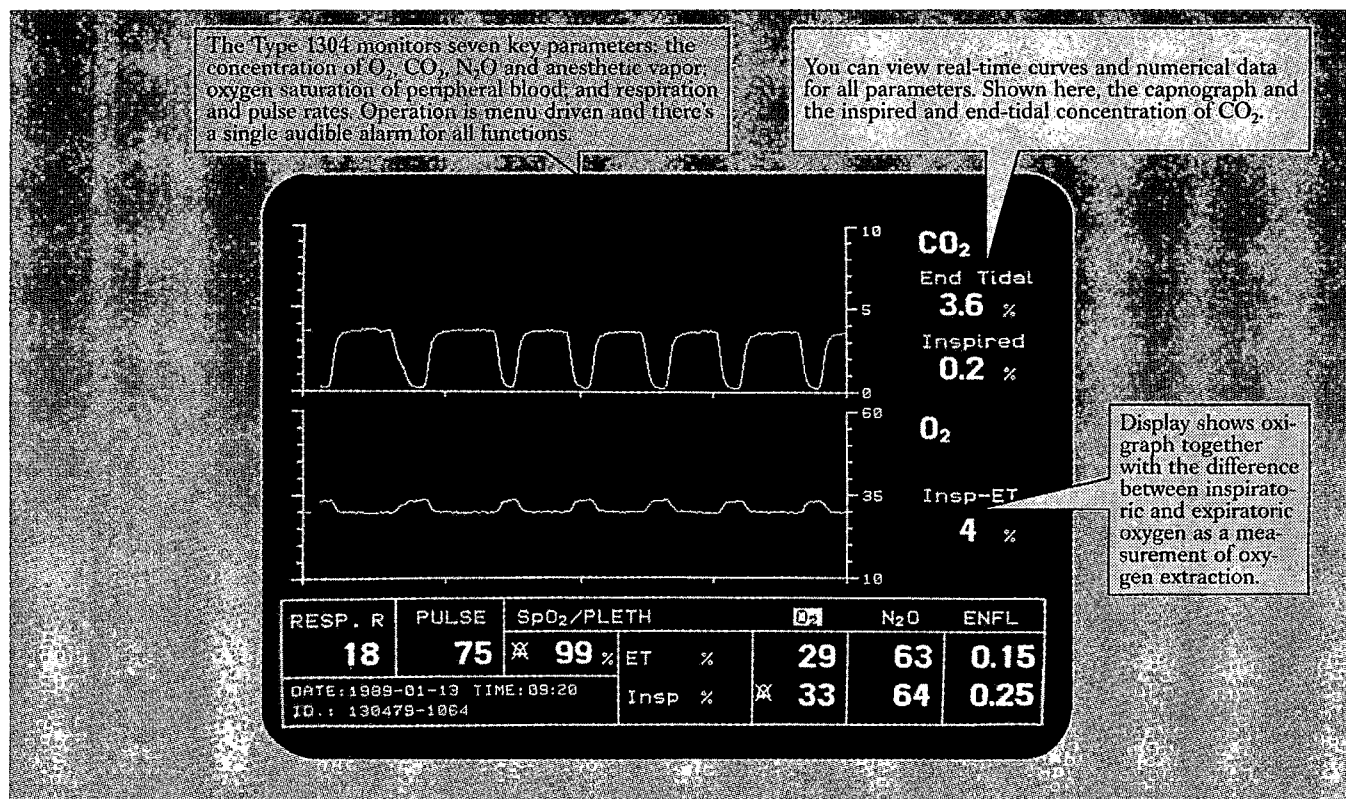
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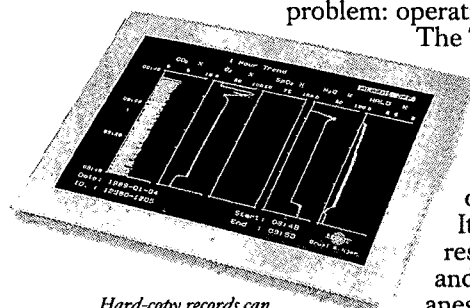


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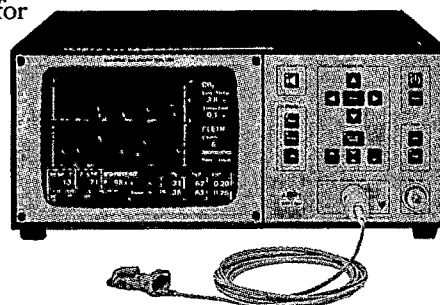
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particularly for periods of up to 3 weeks. To try and balance these points one must ask if they have changed their own policies on the use of epidural analgesia.

Finally, Drs Roberts and Petts (*Anaesthesia* 1990; 45: 376-7) describe a case of meningitis after an obstetric spinal anaesthetic and draw the somewhat breathtaking conclusion that 'the use of spinal anaesthesia in these patients is open to question'. It is a little disappointing that in their otherwise wide ranging consideration of the aetiological possibilities they did not mention the anaesthetist's hands as a possible route to entry of either chemical or bacteriological contamination. The use of a 'no-touch' technique is an essential part of good spinal anaesthetic practice so that such contamination may be avoided. It is of course impossible to say what was the cause in their patient, but all the possibilities must be considered.

These three specific points illustrate a general one. Regional anaesthesia requires not only different knowledge and skills to general anaesthesia, but also a different approach. The anaesthetist must choose and use such methods with care or complications will undoubtedly follow. This can be very demanding. The late Alfred Lee, although an enthusiast for regional techniques, considered that some anaesthetists might not be capable of meeting this demand,¹ but I hope that, in a country where anaesthesia is physician-based, it is something that all can achieve.

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A reply

Thank you for the opportunity to reply. The comments in my editorial were not intended to refer to the combination of regional anaesthesia and 'sufficient sedative or anaesthetic drug to ensure unconsciousness'; awareness cannot occur if consciousness is ensured! My concern relates to situations in which regional anaesthesia is used with small doses of sedative agents, or when no sedation is used and I agree with Dr Wildsmith that careful management is required particularly during Caesarean section. All steps must, I agree, be taken to ensure that patients do not suffer distress during surgery, irrespective of the anaesthetic technique employed. I agree with Dr Wildsmith that the patient should be advised to report any discomfort, that reassurance should be given that discomfort will be relieved and that general anaesthesia should be administered if necessary.

However, I cannot agree with Dr Wildsmith that patients should be warned only of the possibility of discomfort. Pain and discomfort are regarded by patients as very different sensations. Both entirely subjective phenomena, and stimuli which are perceived by some patients as discomfort are interpreted by others as pain. Some experiences during regional anaesthesia will be perceived as

pain by all patients. If patients are warned only that discomfort may occur, the experience of pain is alarming and distressing, and will be recalled after operation even if it is possible to administer sedative or anaesthetic drugs promptly. A portion of patients for a variety of reasons now find pain unacceptable during operations conducted under regional anaesthesia. A warning of the possibility of pain before procedures carried out under regional anaesthesia in circumstances in which intra-operative experiences may be recalled is, in my opinion, advisable not only because distress is likely to be reduced if the experience of pain is known to be a possibility but also because truly informed consent can be given if such a warning is provided.

Failure to supply pertinent information before obtaining consent places the anaesthetist in a potentially vulnerable position if litigation occurs.

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A.R. AITKENHEAD

A reply

We thank Dr Wildsmith for his interest and are grateful for this opportunity to respond to his comments.

Dr Wildsmith admits to some concern about the use of epidural analgesia in some of our patients for long periods of time. We would like to emphasise that those patients who finally died and underwent postmortem epidural examination represent, of course, a negative selection. We do not usually insert epidural catheters in the presence of systemic infections, but sometimes patients develop severe postoperative infectious complications while the epidural is in place. Epidural analgesia was prolonged for up to 3 weeks despite the development of septic complications in some patients, since this management caused excellent analgesia in those patients who had incurable malignant disease. We agree that this should imply a continuous process of decision making, that is: the risk/benefit ratio has to be re-evaluated for every patient day to day.

We discontinue the use of epidural analgesia, as stated in our paper, if a patient with nonmalignant disease develops severe postoperative infectious complications. We check every patient, not only for signs of plasma coagulopathies, but also for thrombocytopenia before the catheter is inserted as a consequence of our findings. We do not recommend the use of epidural anaesthesia in patients with platelet counts below 100 000/cu mm, for example in parturients with HELLP-syndrome.¹ We completely agree with Dr Wildsmith, that the anaesthetist must choose and use such methods with care or otherwise complications will undoubtedly follow.

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E. STRIEPLING

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Anaesthetic research: new approaches need fresh attitudes

Dr Knill, in his excellent editorial (*Anaesthesia* 1990; 45: 271-2) encourages anaesthetists to consider the long-term outcome from anaesthesia. He is obviously well aware of

the sea change in attitudes that will be needed: 'These (postoperative) problems, *although difficult* . . .' (my italics).

The first difficulty is that it is easier to make physiological and pharmacological descriptions than to study clinical outcome. Knill implies that these descriptions may be interesting but it is only the outcome that matters. There are few clinicians who would disagree, but much clinical practice is based on these descriptions.^{1,2}

Secondly, the earliest source of funding for research in anaesthesia, and usually for short-term projects, is from industry, which is unlikely or unable to take much interest in the sorts of question Knill wishes us to ask. Hillman,³ who wrote about intravenous fluids in the peri-operative period, made the observation: 'Finally, and perhaps cynically, there are very few new and expensive fluids being used on a widespread basis, making private funding for research in these days of financial stringency, difficult.'

Not cynically, but realistically: the recent changes in the way universities are funded, and the current obsession with molecular medicine, do not make it easier to find money for research in anaesthesia.

Thirdly, we must abandon the idea that the only worthwhile research is research by individuals or small groups: what might be termed 'first author research'. It would be better for clinicians who generally wish to improve clinical practice, to take a small part in a large research project to attempt to answer Knill's questions than to take a large part in a small project. This means using the tools of epidemiology⁴ and multicentre trials.⁵ Large studies are needed to answer Knill's questions, because adverse outcomes are rare and because there are so many confounding factors.

Fourthly, the structure of academic anaesthesia may not be well suited to these large-scale coordinated projects. Many doctors who take up lectureships or senior lectureships see the posts as short-term staging posts. Nonclinical lecturers or senior lecturers in mathematics or geography are appointed usually with tenure and that is their job: the system gives academic departments long-term stability.

Fifthly, there is a lack of hypotheses,⁶ without which there is the danger of collection of too much information, on the assumption that if enough data are gathered then a picture is bound to emerge. This danger is compounded in Britain by the current politically motivated rush to clinical audit. If we are not careful there will be a rash of ill-thought-out surveys that are no more than retrospective uncontrolled clinical reports. Surveys and audit are worthwhile if they allow us to make the hypotheses.

Rather than an hypothesis, here is a speculation and one that might provide a way for anaesthesia to obtain 'molecular' money. We have concentrated on the techniques of anaesthesia and their possible effects on outcome, and in these studies it is always considered important that groups of patients be well matched. The results are expressed as the numbers of patients who had particular complications and attempts are made to link these occurrences with anaesthetic factors, or to explain them by known, pre-existing factors that put the patients at risk.

Perhaps the *a priori*, deductive, approach is not always best; perhaps we should take the *a posteriori*, inductive, approach and investigate the patients who suffered complications. There may be molecular clues here that will lead us to hypotheses.

But perhaps the truth is that it is all too difficult. Knill's editorial was optimistic; by detailing past successes and leading on to future challenges it was just what the specialty needs in these troubled times. That penultimate sentence, though, included another two words, written perhaps more in hope than in expectation: 'These (postoperative) problems, although difficult, are

undoubtedly soluble . . .' (my italics). These are the words of the philosopher (Godel's theorem aside which, paraphrased, is that all consistent axiomatic formulations of number theory include undecidable propositions; or, that provability is a weaker system than truth⁷) or the optimist, not of the realist. Some clinical problems may for practical purposes be insoluble and it does no harm to admit this, provided that the admission does not lead to undue pessimism or, worse, to nihilism.

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N.W. GOODMAN

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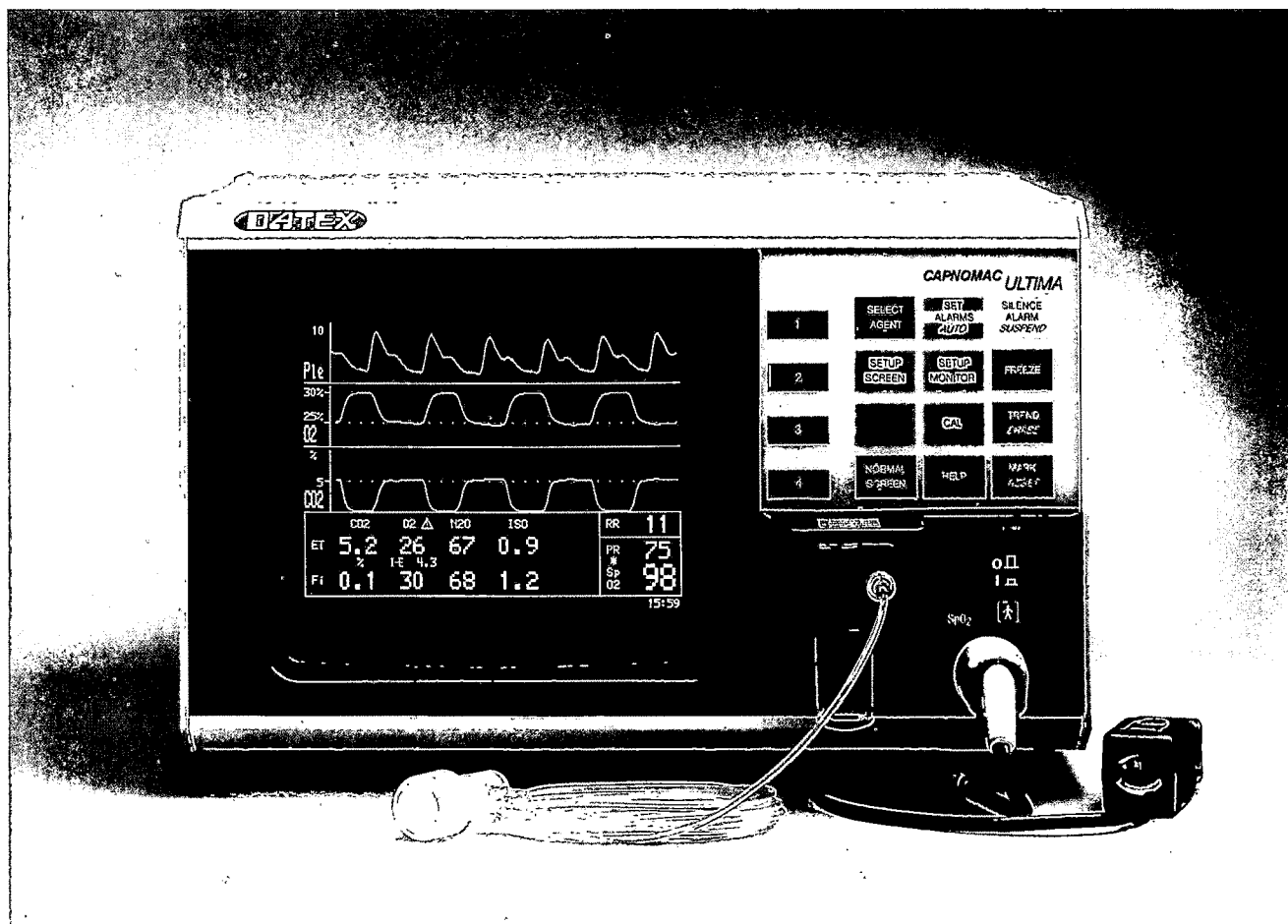
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A reply

Research was called the 'Art of the Soluble'.¹ Researchers see it as their business to solve important problems; they do not wish to waste their time on insoluble tasks. Dr Goodman questions whether the postoperative problems identified in my Editorial are soluble and suggests that this area of research is 'all too difficult'. This is a contention which practical minded researchers will wish to see addressed.

A reason for believing that the postoperative problems of interest can be solved by research was indicated in the final phrase of the sentence to which Dr Goodman refers: 'these problems, although difficult, are undoubtedly soluble, like many faced by anaesthetic researchers in the past' (my italics).² Researchers in anaesthesia faced in the past an enormous array of challenging problems, many of which must have seemed almost insurmountable at the time. These included, for example, the complications of aspiration pneumonia and excessive respiratory depression, and the challenges of the provision of anaesthesia for surgery within the thorax and the skull. These problems, although difficult, were successfully resolved by research that first advanced knowledge about the pertinent physiology or pharmacology and then found ways of prevention or management of the critical physiological or pharmacological events. There is little essential difference in what would be required to resolve the postoperative problems of postoperative myocardial infarction, idiopathic delirium or stroke. Each of these problems occurs at a substantial rate and in a predictable pattern after operation, and is undoubtedly due to definable physiological or pharmacological events. There is no reason to consider any of them inherently insoluble. What will be needed is to understand these events and then to find means of controlling them. There is no basis for believing that researchers cannot achieve these ends with respect to the postoperative problems, as they did with anaesthetic problems in the past.

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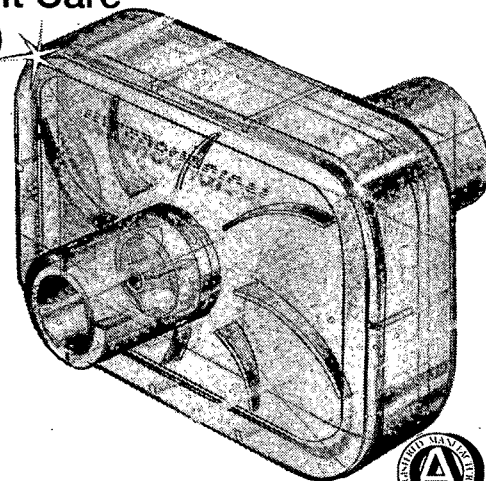
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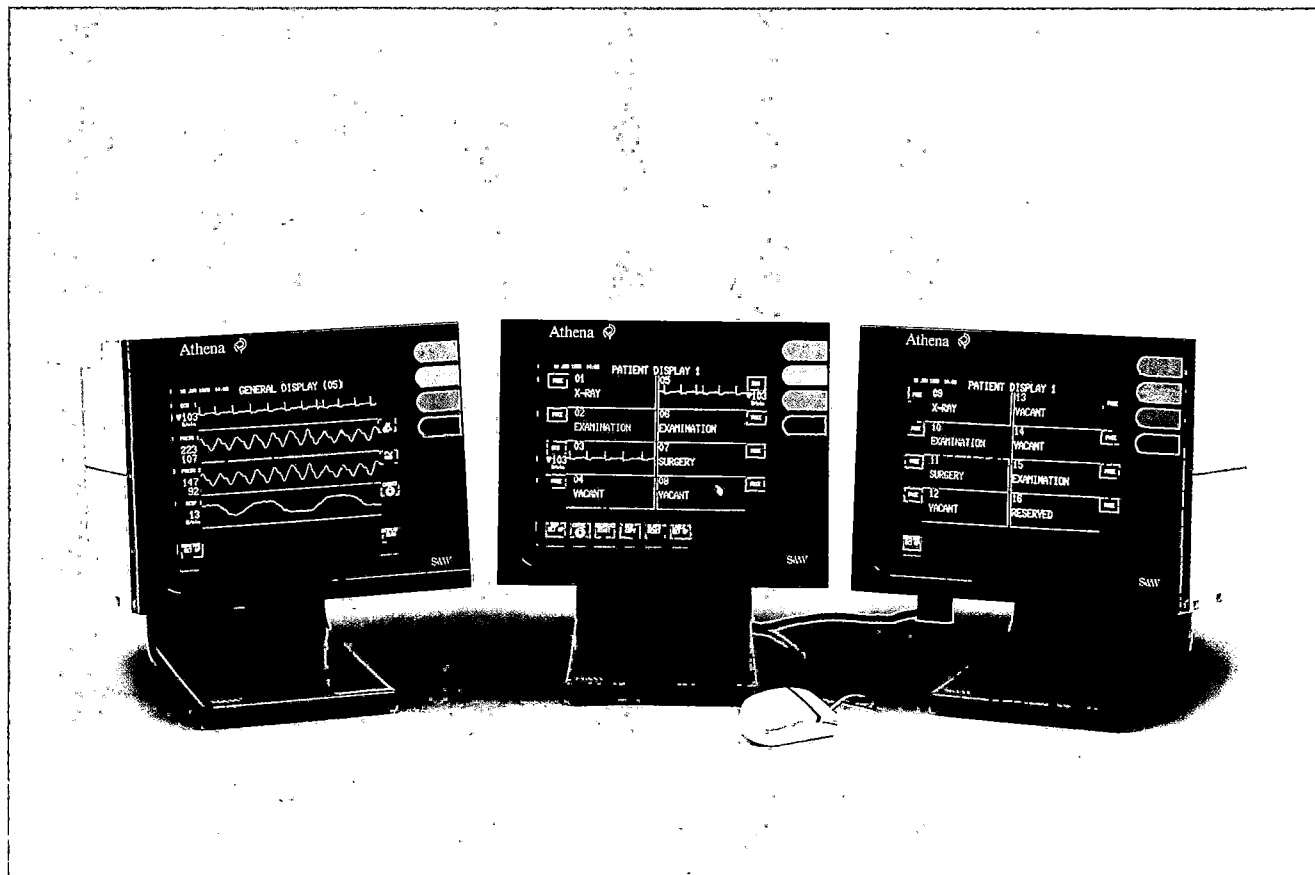
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The several specific hurdles listed by Dr Goodman, the difficulties in changing attitudes and academic structures, in developing hypotheses, and in securing funding, do not constitute impenetrable barriers to getting on with the task! In my experience (which is entirely Canadian), such difficulties are genuine impediments to research only when one proposes to investigate problems that are trivial. The clearly important problems, and I would contend that the postoperative complications in question are the most important unresolved problems of safety in the entire perioperative period, stand an excellent chance of stimulating hypotheses, attracting support, and so on. Dr Goodman's concerns about industrial interests, 'molecular money, and 'first author research' are quite secondary if not irrelevant. (Incidentally, these are the words not of a philosopher but of a practical and considerably experienced investigator).

Sir Herman Bondi, writing in a splendid new book about Newton, suggests that amongst all scientific problems, 80% are insoluble, 19.5% are trivial and only 0.5% are both soluble and important. 'It is the task of the scientist to select the tiny layer between the insoluble and the trivial

where skill, insight and originality . . . can make a difference'.² Dr Goodman suggests that the postoperative complications fall within the large group of problems that are insoluble and, presumably, not suitable for investigative effort. I believe that they lie within the much smaller group that are both soluble and important and that are therefore entirely appropriate for insightful research. Where we can agree, I hope, is that these problems are not amongst the trivial and that they deserve far greater attention than they have received in the past.

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R.L. KNILL

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Unilateral spinal anaesthesia and prolonged femoral nerve block

We wish to report a case of both these problems in the same patient.

A 72-kg male patient was admitted for repair of varicose veins by a left high tie and bilateral multiple ties. He had a history of mild angina, but he suffered no symptoms from his back. He was taking no medication; physical and laboratory examinations were normal.

Premedication was with temazepam 20 mg; a 16-G cannula was inserted into his left hand and he was turned onto his right side. A 25-G Spinocan needle was inserted at L_{3/4} after a right paramedian approach. Clear cerebrospinal fluid (CSF) flowed into the hub and 2.5-ml bupivacaine 0.5% heavy was injected into the subarachnoid space. Clear CSF was aspirated after injection. The patient was immediately returned to the supine position. A dense unilateral motor and sensory block to T₈, as tested by pinprick and ethyl chloride spray, was evident in the left, nondependent side at 5 minutes. There was no block on the right. The patient was positioned on his right side with slight head-down tilt for a further 15 minutes. There was no change in the block achieved at 20 minutes: sensation and power in the right leg were normal.

General anaesthesia was then induced with thiopentone 250 mg and maintained with nitrous oxide, oxygen, enflurane delivered through a Bain system and mask.

A right femoral nerve block was carried out using a 22-G regional block needle to inject 15 ml 0.5% plain bupivacaine. No resistance to injection was noted after careful aspiration.

Complete loss of power and sensation on his left leg and loss of power and sensation in the territory of the right femoral nerve were noted on recovery from the general anaesthetic.

Twenty-four hours later we were asked to review the patient who gave a history of weakness of the right leg; he had fallen twice during attempted mobilisation.

Examination revealed residual sensory loss in the territory of the right femoral nerve with corresponding loss of power in the quadriceps (gauged 4/5). The left leg was normal. The patient was able to mobilise at 30 hours after operation. Power was now 5/5 in the right quadriceps but a small oval patch of anaesthetic skin 20 × 10 cm was present over the anterior thigh. The right leg was neurologically normal by 40 hours.

We accept that the use of a single shot technique in an anaesthetised patient for femoral nerve block may result in inadvertent intraneural injection or trauma caused by the needle. Inadvertent puncture of the femoral artery with haematoma formation seems unlikely in the face of negative aspiration tests.

We suggest the existence of a septum dividing the subarachnoid space in two in this patient, to explain the unilateral block on the nondependent side, and we assume the needle must have entered the upper compartment.

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C.N. BARTHRAM

Meningitis after spinal anaesthesia

Drs Roberts and Petts draw attention to a case of meningitis after obstetric spinal anaesthesia (*Anaesthesia* 1990; 45: 376–7). They do not mention the use of a filter.

It is our routine to draw up all drugs used for spinal

anaesthesia through an epidural filter. The 0.22- μ m pores prevent the injection of bacteria and particulate matter.

It is impossible to state that this would have prevented the meningitis in their case, but we believe that the use of

such filters should be mandatory for spinal anaesthesia; after all, we always use filters for epidurals.

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S. McHALE
M.M. CLARK

A reply

The use of filters for epidural anaesthesia is a well established practice in the United Kingdom. The use of filters for drawing up agents for spinal anaesthesia is, however, less widespread at present. James *et al.*¹ showed that the Millex filter reduced contamination from syringes which were refilled several times over a period of hours during obstetric epidural anaesthesia. The drawing up, under sterile conditions, of a 'single shot' dose of local anaesthetic agent is hardly comparable to this situation. Crawford² highlighted the dilemma about all sterile

precautions for obstetric regional anaesthesia when he contended that, although the evidence that filter use is effective is inconclusive, a filter seems a relatively inexpensive and reasonable precaution. It is as yet unproven whether the use of filters reduces the incidence of meningitis after spinal anaesthesia and it would seem precipitate to consider their use to be mandatory. The use of filters, although protective against bacterial and particulate contamination, would not prevent chemical contamination of the cerebrospinal fluid.

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Continuous epidural infusion

The paper by Lamont and colleagues (*Anaesthesia* 1989; **44**: 893-6) was interesting and it confirmed the efficacy of a continuous epidural infusion of 0.125% bupivacaine at 10 ml/hour in labour.

Midwives do indeed need to spend less time recording serial blood pressure results. However, the authors' conclusion that 'the regimen is likely to be much less labour intensive' may be taken by some to imply that these patients can safely be left alone during the long periods between top-ups. This assumption should be firmly discouraged, since the possibility of transdural migration of the epidural catheter, with subsequent extension of the block and hypotension, must be remembered with this technique. No patient with an epidural in progress should be left unattended and, if a continuous infusion is used, the height of the block should be assessed at least every 2 hours.¹

New developments in anaesthetic practice must be introduced with a zealous regard for safety; this is particularly true in the field of obstetric analgesia, which is especially sensitive to the adverse publicity associated with any outcome other than perfection. The use of a continuous infusion of epidural local anaesthetic in labour requires, if anything, more vigilance than the traditional method.

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Nottingham NG5 1PB

D.G. BOGOD

Reference

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Race and Apgar scores

Dr Virginia Apgar introduced a new neonatal scoring system in an article entitled 'A proposal for a new method of evaluation of the newborn infant' in 1952. Each of the five variables, heart rate, respiration, muscle tone, reflex activity and skin colour, was given a score of 0, 1, or 2, for a maximum score of 10. The Apgar score became within 10 years a routine part of initial neonatal assessment in most parts of the world. An acronym, Apgar, was proposed in order to remember the constituents of the scoring system: appearance or colouring, pulse, grimace or reflexes, activity as judged by muscle tone and respiration.¹

An Apgar score of 7 to 10 indicates that the infant's condition is stable, 4 to 6 corresponds to moderate asphyxia and 3 and below indicates severe asphyxia. Resuscitation measures depend on the Apgar score.²

We observed that the Apgar score, in general, is lower in healthy Afro-American neonates than in their Caucasian counterparts. A retrospective analysis of Apgar scores in 544 Caucasians and 51 Afro-American new born infants were undertaken in order to test this hypothesis. All neonates were healthy and full term. Umbilical cord arterial and venous blood gases were normal in all cases. All infants with congenital anomalies were excluded. A

Table 1. 1- and 5-minute Apgar scores in Caucasian and Afro-American Neonates. Mean Apgar score (SD).

Race	1 minute	5 minutes	p
Caucasian	8.6 (0.58)	9.08 (0.38)	<0.001
Afro-American	6.7 (1.47)	8.57 (0.76)	<0.005

Wilcoxon nonparametric test was used to determine whether or not there is a significant difference in mean Apgar scores at 1 and 5 minutes (Table 1).

Thus, healthy Afro-American infants have lower Apgar scores than their Caucasian counterparts. The component of the score responsible for this difference is probably the colour of the skin. It is very unusual for an Afro-American to be completely pink³ at 1 and 5 minutes after birth. The optimal Apgar score for an Afro-American infant should be therefore lower.

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- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

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CONTENTS

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Low cardiac output and enoximone

Dr White's account of the management of low cardiac output syndrome after cardiac surgery with enoximone (*Anaesthesia* 1990; 45: 386-89) intrigued me.

The residual aortic regurgitation after mitral valve replacement and coronary artery bypass grafting was presumably sufficiently serious to contraindicate the use of intra-aortic balloon counterpulsation and yet so trivial as not to justify replacement.

Secondly, I was astounded to see the use of adrenaline at an infusion rate of 2.5 ($\mu\text{g}/\text{kg}$)/minute. This seems to be approaching the dose range for a single injection in the treatment of anaphylaxis, and well beyond the maximum useful inotropic dose quoted as 0.2 ($\mu\text{g}/\text{kg}$)/minute.¹

The systemic vascular resistance (SVR) was calculated and I presume not indexed, but no mention was made of its inaccuracy due to the presence of (important) aortic regurgitation. Quite clearly the run-off after the left ventricle had ejected its stroke volume would be forwards into the systemic arterial tree and backwards through the aortic orifice into the left ventricle. Thus the mean arterial pressure would be spuriously low for the purpose of calculating SVR. The merit of having a low SVR in the presence of aortic regurgitation is obvious but disadvantageous in some ways for effective coronary perfusion.

How much enoximone contributed to the increase in cardiac index and decrease in calculated SVR when measured after 22 hours seems a matter of conjecture. The happy outcome for the patient in this anecdotal report might even be entitled 'successful management of the circulation using metaraminol'!

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Southampton, SO9 EXY

J.W. MANNERS

Reference

1. HENSLEY FA, MARTIN DE. *The practice of cardiac anesthesia*. Boston: Little, Brown, 1990.

A reply

We welcome the opportunity to reply to Dr Manners' letter. Intra-aortic balloon counterpulsation was contraindicated in this patient because of the tachyarrhythmia rather than the aortic regurgitation, which was considered to be of minor significance. Triggering of the intra-aortic balloon pump from the ECG signal produces inconsistent pump responses in tachyarrhythmias and makes it ineffective.¹

The systemic vascular resistance figures we quoted were not indexed. We considered that the reduction in SVR with enoximone was much greater than could be explained by the degree of aortic regurgitation alone.

Flow improved considerably after enoximone but sufficient driving pressure must be maintained to perfuse vital organs, and a balance between pressure and flow must be struck. Metaraminol has been used regularly as the primary vasopressor at Papworth for over 20 years. Like many drugs, it is the way in which they are used that is important.

We would also be astounded by the use of adrenaline at 2.5 ($\mu\text{g}/\text{kg}$)/minute and must thank him for pointing out the obvious typographical error! The adrenaline infusion should read 0.25 ($\mu\text{g}/\text{kg}$)/minute.

Case reports are by their very nature anecdotal; however, we considered the case worth a report. This was our first experience with a new class of inotrope in 1987 and we learned several lessons.

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D.A. WHITE
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Occupational exposure to HIV and zidovudine chemoprophylaxis

Comprehension of risk of HIV infection through normal working exposure to the virus in theatre and intensive care comes largely from material intended for the lay public. Anaesthetists, however, work in an environment where they are regularly exposed to blood and body fluids and where needlestick injury is not uncommon.

The risk depends on the prevalence of HIV in the population, probability of transmission per accident and the number of accidents in a given time. There are 3021 recorded cases of HIV in the United Kingdom; estimates for the number of unidentified carriers ranges from 25-62 for every known AIDS case. Large scale studies of prevalence are difficult, but a recent study of over 115 000 pregnant women, tested anonymously by use of Guthrie cards for neonatal screening, has shown a figure as high as 0.49 per thousand in inner London, in a group which is not essentially high risk.¹

The incidence of needlestick injury and risk of subsequent transmission is unknown. Combined data from 10 prospective studies shows that the risk of HIV-1 transmission associated with a single parenteral exposure is 0.37% (slightly less than 1 infection for every 250 exposures).² This is probably an underestimate because inoculation with small numbers of viruses, as in needlestick injury, can result in delayed seroconversion and a delay of 42 months is recorded.³

Any one exposure would seem not to pose an unacceptable threat, but the cumulative risk of many incidents may be unacceptably high. Jones³ has suggested that the chance of infection for an anaesthetist over a 40-year working life is as high as 1 in 25. The risk of progression to AIDS is high once infection has occurred.

The Association of Anaesthetists of Great Britain and Ireland has published guidelines for the prevention of

infection⁴ and while adherence to the guidelines will reduce the risk they will not eliminate it. This is widely recognised and there is at present interest in chemoprophylaxis with zidovudine after exposure.

Zidovudine is the only drug with proven *in vivo* efficacy against HTLV III in humans and has been shown to alter the course of animal retroviral infections if given immediately after exposure.⁵ Several institutions now offer zidovudine prophylaxis after contamination.

Such use is controversial, and doubts remain about both its efficacy and toxicity. Zidovudine interferes with the action of viral RNA-dependent DNA polymerase (reverse transcriptase). It is a potent inhibitor of replication, so it seems unlikely that this drug could prevent infection unless it were administered during the initial replicative cycle. Moreover, proving efficacy is difficult because of the low incidence of infection after each exposure.

Nor is the toxicity in healthy people known. The major acute problems in AIDS patients are granulocytopenia and anaemia which are dose-dependent and reversible. They rarely occur in the first month of therapy and so serious toxicity is unlikely with short course prophylaxis. However, since zidovudine is active at the level of DNA polymerase enzymes, it may be teratogenic and affect reproductive capacity. Additionally, in studies conducted by the manufacturers, vaginal carcinomata were observed in mice which received doses that the Federal Drug Administration considered produced levels approximately equal to human plasma levels.⁶ Thus, zidovudine may also be carcinogenic in humans.

Anaesthetists must realise they are in a risk group for AIDS and observe rigorously the infection control measures outlined by the Association. It is important to be

aware zidovudine chemoprophylaxis exists, that it may be appropriate after high risk exposure, and that it must be instituted as soon as possible after exposure. This should be balanced with the low risk of infection, the question of efficacy, and the unknown short term and long term toxicity.

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N.G. SMART

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Hyperkalaemia, cardiac arrest, suxamethonium and intensive care

We report a case of hyperkalaemia and associated cardiac arrest after the administration of suxamethonium to facilitate a change of tracheal tube.

The patient was a previously fit 55-year-old man who suffered a cardiac arrest whilst at work. He was resuscitated by a passer-by and admitted to this hospital with a good cardiac output but in ventilatory failure from a flail segment sustained during the resuscitation procedure.

He was admitted to Intensive Care for controlled ventilation of the lungs. He became pyrexial and developed left midzone shadowing on his chest X ray over the next 48 hours. His respiratory function subsequently deteriorated and he became dependent upon high-inspired oxygen concentrations and positive-end expiratory pressures to maintain adequate oxygenation. We were unable to determine either the cause of the cardiac arrest or the subsequent respiratory disorder despite extensive investigation. His condition failed to respond to antibiotics. His cardiovascular system was stable, and biochemical investigation showed only minor degrees of hepatocellular and renal dysfunction. Nutrition was administered parenterally once it was evident that his admission was likely to be protracted.

His tracheal tube was changed three times during the first 18 days of his intensive care admission and on each occasion he was given suxamethonium 100 mg to provide neuromuscular relaxation. He suffered no untoward reaction on any of these occasions.

Tracheostomy was performed on day 20. The diagnosis was unknown. The working diagnoses were pulmonary embolism or a vasculitic process, therefore the patient was fully anticoagulated with heparin and given methyl

prednisolone 500 mg daily for 10 days. The patient's respiratory function subsequently improved, sedation was discontinued and weaning started.

Thirty-one days after admission he suffered a haemorrhage from an area of tracheal ulceration secondary to cuff trauma from a standard size 9.0-mm Portex tracheostomy tube with a low pressure cuff. A special tracheostomy tube, 3 cm longer than standard at the patient end, was manufactured by Portex. The patient was confused, somewhat uncooperative and coughed violently with any manipulation of the tracheal tube. It was therefore decided that a short general anaesthetic was appropriate to facilitate insertion of the longer tracheostomy tube and to minimise further trauma to the tracheal mucosa.

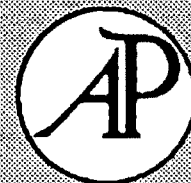
The patient was pre-oxygenated, and propofol 160 mg and suxamethonium 100 mg were administered. The tube change was uneventful with no period of hypoxia, and mechanical ventilation was re-established. Approximately 90 seconds later the ECG monitor showed sinus bradycardia which was unresponsive to 0.6 mg atropine. Ventricular tachycardia and then ventricular fibrillation occurred. External cardiac massage was started and the patient's lungs were ventilated with 100% oxygen.

The rhythm was unresponsive to DC cardioversion with 200, 300, and 400 joules. Lignocaine 100 mg and subsequent cardioversion was also unsuccessful. The patient then became asystolic with no response to atropine 1.2 mg, calcium chloride 100 mmol and adrenaline 1 mg. Arterial blood gas analysis, taken approximately 5 minutes after the cardiac arrest, showed a serum potassium of 8.7 mmol/litre which was confirmed on a second specimen.



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(The patient's serum K^+ = 3.7 mmol/litre, urea = 11.6 mmol/litre and creatinine = 55 μ mol/litre that morning). Repeated doses of calcium and adrenaline produced ventricular tachycardia which reverted to ventricular fibrillation with a single 200 J, DC shock. A total of 30 ml 10% calcium chloride and 1 g magnesium were administered to treat the hyperkalaemia. The cardiac rhythm rapidly reverted to a junctional tachycardia and then sinus tachycardia with a good cardiac output. Insulin and dextrose therapy were then instituted and continued until a serum K^+ less than 5 mmol/litre was obtained. Forty-five minutes after cardiac arrest the serum K^+ = 5.1 mmol/litre.

The patient was sedated and the lungs ventilated overnight. There was no evidence of acute myocardial infarction after serial ECG and cardiac enzyme examination.

Suxamethonium is well recognised to cause a significant increase in serum potassium in patients with burns, paraplegia, denervation syndromes and renal failure, but the patient reported here did not fall into any of these categories. There has been only one previous report of hyperkalaemic cardiac arrest in ITU associated with suxamethonium.¹ This occurred in a septic patient after 17

days in ITU. The only evidence of infection in our patient was a pyrexia. He had no leucocytosis, was not septicemic and there was no microbiological or serological confirmation of infection.

The patient had been in ITU for 34 days when he suffered this event. This unusual reaction to suxamethonium may be related to his lengthy ITU stay which caused disuse atrophy of his muscles. This atrophy may have been exacerbated by his course of methylprednisolone. There are, however, no reported associations between glucocorticoids and suxamethonium-induced hyperkalaemia.

The patient subsequently made a good recovery and has since returned to work. The primary diagnosis remains a mystery.

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Atracurium in whole body hyperthermia

Little is known about the effectiveness of atracurium in patients suffering from hyperthermic infections or whole body hyperthermia.¹

We report the case of a 30-year-old man who underwent whole body hyperthermia using cardiopulmonary bypass. Blood gas tensions and serum electrolytes were checked at 30-minute intervals throughout the procedure and corrected according to the actual temperature. The patient was anaesthetised with thiopentone and fentanyl and ventilated mechanically (P_{aCO_2} 4.6 kPa). Neuromuscular transmission was assessed with a peripheral nerve stimulator (Myograph 2000 Biometer). Atracurium 0.25 mg/kg (ED_{90}) was administered in the normothermic state after a baseline neuromuscular response was established. Onset and duration of relaxation was within normal range (Table 1, A). The same dosage of atracurium was given again in the hyperthermic steady-state and a prolonged onset, decreased intensity and short duration of relaxation was found (Table 1, B). Vecuronium 0.05 mg/kg

(ED_{90}) was administered after spontaneous recovery (T1 100%, ToF 96%). The neuromuscular blockade was normal despite the high temperature (Table 1, C).

This abnormal response to atracurium could be explained by increased metabolism during hyperthermia from high tissue perfusion or to an increased distribution volume due to extracorporeal bypass. We found a normal response to an equipotent dose of vecuronium under the same conditions.

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Reference

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Table 1.

Drug	Core temperature (°C)	T1 depression (%)	T1 onset (minutes)	Duration to 75% (minutes)
A, Atracurium (0.25 mg/kg)	37.0	100	3.2	22.5
B, Atracurium (0.25 mg/kg)	41.5	46	4.2	13.7
C, Vecuronium (0.05 mg/kg)	41.5	100	2.5	25.8

HMEs and body temperature

Drs Ip Yam and Carli (*Anaesthesia* 1990; 45: 563-5) have demonstrated that one type of heat and moisture exchanger is not effective in preventing a decrease in body temperature. Their findings should not be generalised to all such devices.

The principle of the heat and moisture exchanger was invented in 1836 by Julius Jeffreys MRCS, FRS (1800-1877).¹ He based the design of his Respirator on it, a

device which was intended to warm and humidify the inspired air and so relieve the cough of people suffering from chronic bronchitis. Wearers of the Respirator soon observed that it possessed 'in a powerful degree, the property of diffusing warmth over the whole system'. Jeffreys 'met with persons describing of their own accord this general warming effect as a source of comfort; and, what is curious, that it answered the purpose of a great

coat.' Jeffreys's explanation was that a very much smaller quantity of heat was 'lost by the lungs in respiration, partly in raising a cold air up to blood heat, and in part in the form a latent heat carried off in the vapour exhaled.'²

Jeffreys, who was no mean physicist, recognised, from a consideration of the physical principles involved, that for efficient heat transfer both a good conductive material and a cascade mechanism were needed. His heat and moisture exchanger consisted of from eight to 12 closely woven lattices of very fine silver wire, each insulated from the next.³ He took the unusual step of patenting it in his concern to ensure the accurate manufacture of the device, and to protect it from less effective imitations.

Mapleson and his colleagues, in their investigation of condense-humidifiers, supported, albeit unwittingly, Jeffreys's stipulation that for maximal effect the lattices, or gauzes, should be thermally isolated from one another, and

about 10 layers thick.⁴ The limitations of the Portex device, therefore, argue against its failure to conform in its design to the principles established by Jeffreys.

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Inhaled opiates

The 'recent interest' in the delivery of opiates by nebuliser (*Anaesthesia* 1990; 45: 449) is rather longstanding. Beigel¹ wrote his treatise in 1866 'On Inhalation'. This deals with the application of nebulised solutions in the treatment of various chest diseases, which was a novel approach at the time designed to get the drug to the site of action.

He, (although not the beneficiary of our present knowledge of pharmacokinetics) comments 'It must be borne in mind that besides the local effect of the spray, absorption takes place also, and that medicaments are even much more readily absorbed through the mucous membranes than they are by internal application—a fact which must be taken into consideration when the dose is to be decided upon.'

The pharmacopoeia of suggested drugs for inhalation is rather limited, but it includes the following: '9. *Tincture of opium*, from five to twenty minims (approximately 3–12 mg morphine) to one ounce of water, is very often beneficially applied, when it is our aim to rid the patient of a troublesome cough, be it a symptom of phthisis or of any other pathological process of the respiratory organs, provided that no phenomenon exists preventing the application of opium in general.'

It will be interesting to observe how the story unfolds this time around, and I can do no better than to close with the end of Beigel's treatise. 'He who expects wonders from that mode of treatment (nebulised drugs) will soon be disappointed: he who recommends it as an infallible one, will prove a false prophet; but an unprejudiced application of the atomizer will lead to a conviction that the invention of Sales-Girons has been a most valuable addition to therapeutics.'

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Reference

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A reply

We would like to thank Dr Harrison for drawing attention to the historical aspects of our paper. Interest in the delivery of opiates in this manner is recent in the anaesthetic literature, but the concept is indeed a very old one as Dr Harrison points out. Opiates were used by religious cults in the Eastern Mediterranean area as early as 5000 years ago,¹ and an opium pipe dating from the 12th century BC found in Cyprus² indicates that their administration by inhalation is at least 3000 years old.

We agree entirely with Dr Harrison that in general terms the technique is not new, and also with his 19th century source Beigel, in that we should not have unrealistic hopes of any treatment until it has been thoroughly investigated.

To this end, we hope our application of a pharmacokinetic and pharmacodynamic approach to the use of fentanyl in this manner is a step along that path.

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Oximetry in pulseless disease

We report the use of a pulse oximeter in two patients with pulseless disease to monitor arterial oxygen saturation and systolic blood pressure.

A 30-year-old female, who was known to have aorto-arteritis presented with chronic suppurative otitis media for mastoidectomy under general anaesthesia. Examination revealed bilaterally weak carotid pulsations and absent pulses in both the arms. The pulses in the lower limbs were

palpable, and blood pressure of 140/96 mmHg was recorded.

A pulse oximeter (Ohmeda Biox 3700) was used to monitor the systolic pressure in the upper limb. The finger probe of an oximeter, when placed on the right hand, showed a distinct plethysmographic waveform with oxygen saturation (SpO₂) of 92–95%. The pulse oximeter was judged to be functioning accurately once the pulse rate

displayed by the oximeter and cardioscope corresponded. An appropriate size blood pressure cuff connected to a mercury manometer was placed on same arm. The cuff was manually inflated in 2–5-mmHg increments till the plethysmographic waveform disappeared and the reading recorded. The cuff was then inflated to 250 mmHg and gradually deflated until the waveform reappeared on the oximeter and the monometer reading recorded at this point. The average of the two recordings was taken as systolic blood pressure in the upper arm.

This technique to measure the systolic blood pressure was adopted in the intra-operative and postoperative periods.

A 45-year-old male, known to have aorto-arteritis presented to the ICU in grade IV coma and with irregular breathing (rate 44/minute). All the peripheral pulses and left carotid pulse were absent. Therefore, measurement of systolic blood pressure by conventional techniques was not possible. Management included ventilatory support and general supportive care. The pulse oximeter was used to

monitor systolic blood pressure (as described above) during his 9-day stay in the ICU.

It is technically difficult to obtain reliable estimates of systolic blood pressure in these patients. Ramanathan *et al.*¹ observed a pulsatile blood flow in clinically nonpulsatile arteries in patients with Takayasu's syndrome. We too observed the plethysmographic waveform on the oximeter in the limbs with weak or absent pulses.

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Noncardiogenic pulmonary oedema after attempted suicide by hanging

Acute pulmonary oedema after an attempted suicide by hanging was reported earlier,¹ but it was not defined by the recent diagnostic criteria for adult respiratory distress syndrome (ARDS).² This is a case report of a patient who could not be saved despite best possible supportive care and monitoring where these criteria were used.

A 25-year-old woman was brought to the casualty department of our Institute within 25 minutes of rescue by her family from an attempted suicide by hanging. The exact duration of hanging was not known. She was unconscious with normal sized pupils that were sluggishly reacting to light. There was dusky cyanosis in the peripheries; her respiratory rate was 22/minute. Her arterial blood pressure was 80/60 mmHg and her heart rate 140 beats/minute. There was no evidence of upper airway obstruction but there were crepitations at both lung bases. There was an ecchymotic mark over the anterior aspect of the neck. Her trachea was intubated with a 7.5-sized Portex cuffed nasotracheal tube and manual ventilation of the lungs was started with an Ambu bag with 100% oxygen. This resuscitation improved her arterial blood pressure to 110/70 mmHg. The patient was transferred to the respiratory intensive care unit (RICU) and connected to a Cape 2600 intensive care ventilator with F_{IO_2} of 1.0. Pink frothy secretions were observed through the transparent tracheal tube within a few minutes. The airway pressure was 4.5 kPa. Hypoxaemia (P_{aO_2} 5.7 kPa) and metabolic acidosis was demonstrated on arterial blood gas analysis (sample 1). Furosemide 60 mg, 100 mmol $NaHCO_3$ were given intravenously and 1.0 kPa PEEP was applied. Improvement (sample 2) resulted. A chest radiograph showed pulmonary oedema with densities in all four lung quadrants. A radiograph of the neck was normal. A bedside two-dimensional echocardiograph revealed normal ventricular size, normal sized pulmonary veins, fair left ventricular function and normal valves. 12-lead electrocardiography (ECG) revealed sinus tachycardia. Four hours after admission to RICU, there was systolic hypotension (70 mmHg) which failed to respond to a fluid challenge. PEEP was decreased to 0.6 kPa and a dopamine infusion was commenced at a rate of (5 μ g/kg)/minute which resulted in improvement of systolic pressure to 110 mmHg. The patient regained consciousness the next morning (approximately 8 hours after admission to RICU) with no apparent neurological deficit. She continued to

receive dopamine infusion at a rate of 5 to 8 (μ g/kg)/minute to maintain her systolic blood pressure between 90 to 100 mmHg. The chest X ray was not changed. Arterial P_{aO_2} ranged between 22 and 24 kPa (samples 4, 5 and 6) with an F_{IO_2} of 0.8 and PEEP 0.6 kPa. Cardiac arrest occurred on the morning of the third day from when she was resuscitated. The F_{IO_2} was increased to 1 and dopamine infusion rate to 12 (μ g/kg)/minute after this episode. Sample 7 (after cardiac arrest) revealed a P_{aO_2} of 8.3 kPa. Bedside two-dimensional echocardiography again showed no regional wall motion abnormalities, mild diastolic dysfunction, normal function of the valves, and overall good left ventricular function. Sinus tachycardia continued. PEEP was increased to 1.0 kPa since an F_{IO_2} 1.0 had failed to improve arterial oxygenation (sample 8). Severe pulmonary oedema persisted on X ray. She was resuscitated after five more episodes of cardiac arrest, but she succumbed on the sixth occasion. Monitoring during management in RICU consisted of continuous ECG, direct arterial blood pressure, inspired oxygen concentration, end tidal CO_2 , central venous pressure and urine output monitoring. Intravenous fluids (crystalloids) were administered according to CVP, arterial blood pressure and urine output.

This case illustrates the occurrence of ARDS after an attempted suicide by hanging. The recent expanded criteria² for ARDS using chest X ray, hypoxaemia and PEEP scores were used (see Table). Cardiac failure was excluded by use of bedside two-dimensional echocardiography.

The pathogenesis of pulmonary oedema appears to be similar to that after relief of acute airway obstruction.³ Acute upper airway obstruction during hanging results in a large increase in subatmospheric pressure which may be transmitted to the interstitial peribronchial and perivascular spaces; this disrupts the integrity of the pulmonary capillaries. The high subatmospheric transpulmonary pressure would enhance venous return, while simultaneously fluid therapy would increase the preload and consequently the pulmonary vascular pressure. These cardiovascular haemodynamics are opposed during expiration by the relatively high positive transpulmonary pressure. The expiratory component of acute upper airway obstruction thus acts as a form of retard, akin to PEEP, which exerts a protective effect in the patient. The haemodynamic consequences produced by an abrupt

Table 1. Arterial blood gas analysis and lung injury scores.

Sample	Time	F _{IO₂}	PEEP kPa	pH	P _{aO₂} kPa	P _{aCO₂} kPa	Base excess mmol/litre	Saturation %	X ray score	Hypoxaemia score	PEEP score	Lung injury score
1	Day 1	1	—	7.2	5.7	5.3	−7.5	71	—	—	—	—
2	Day 1	1	10	7.37	27.9	4.0	−9	99.6	4	2	2	2.6
3	Day 1	1	6	7.5	29.0	3.6	1.5	99.6	—	—	—	—
4	Day 2	0.8	6	7.4	23.4	4.5	1	99.6	4	2	1	2.3
5	Day 2	0.8	6	7.4	22.6	4.5	−0.7	99.5	—	—	—	—
6	Day 2	0.8	6	7.4	23.9	4.0	0	99.5	—	—	—	—
7	Day 3	0.8	6	7.0	8.4	7.6	−14	85.1	—	—	—	—
8	Day 3	1	6	7.3	6.2	5.7	−5	71	4	4	1	3
9	Day 3	1	10	7.3	11.8	4.9	−7.1	95	4	4	2	3.3

Chest X ray score, a score of 4 is given if alveolar consolidation is present in all four quadrants.

Hypoxaemia score, a score of 2 is given if P_{aO₂}/F_{IO₂} ratio is 175 to 224 and 4 when it is less than 100.

PEEP score, a score of 1 is given if PEEP applied is 0.6–0.8 kPa and 2, if it is 0.9–1.1 kPa.

ARDS is diagnosed when lung injury score (obtained by dividing the aggregate sum by the number of components (3)) exceeds 2.5; mild to moderate lung injury is said to be present if the score ranges from 0.1 to 2.5.²

decrease in airway pressure, caused by relief of acute airway obstruction without a positive expiratory pressure, might produce a sudden increase in venous return due to an acute redistribution of intravascular volume from periphery to pulmonary circulation. The resultant acute increase in the pulmonary hydrostatic pressure, in the presence of damaged pulmonary microvasculature, leads to pulmonary oedema. Acute hypoxaemia and excessive sympathetic discharge may also contribute.

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Pre-induction behaviour in children

We read with interest the article which reviewed the use of sedatives as preliminary medication for paediatric surgery (*Anaesthesia* 1990; **45**: 427–35) by Morgan-Hughes and Bangham. Their article does not aim to present preparation before operation other than the administration of sedatives, but we think that drowsiness or sleeping before anaesthesia is not usually necessary. Psychological preparation is more important and the emphasis should be on pre-induction calmness.

We aim specifically to have a calm, but awake and cooperative child, before anaesthesia in a child aged more than 3 years. Our technique involves the use of small doses of oral diazepam (0.2–0.3 mg/kg), but with much emphasis on psychological preparation of the child and parents. The child and parents are seen on the day before surgery and a rapport set up with the child. An in-depth knowledge of children's television and magazine characters is helpful, plus an ability to talk with enthusiasm about the child's interests. In conversation with the child the anaesthetist's sympathetic and friendly attitude is projected so that his or her face and voice is remembered. The parents are allowed to gain confidence in the anaesthetist, particularly by the anaesthetist showing them that their child is an important patient. An unhurried attitude is necessary.

The drawing of a picture by the child is discussed and a topic for the picture decided. The child, often full of enthusiasm, will bring a picture that has been drawn the

previous afternoon or evening to theatre to show the anaesthetist. However, the main use of drawing is to occupy the child whilst he or she waits to be anaesthetised. Crayons, paper and a clip board are available in the children's theatre and the child draws a picture of choice, which then joins the many others hanging on the anaesthetic room wall. The child will go into a world of imagination virtually oblivious to the threatening environment of the anaesthetic room.

EMLA cream is placed on the nondominant hand 90 minutes before surgery and an intravenous cannula can often be sited whilst the child continues drawing. The presence of a parent in the anaesthetic room is almost mandatory with the younger child. The child will sometimes cry after insertion of the cannula, but will usually be calm up to this point.

The main ingredients for success are a sympathetic child-orientated anaesthetist, a relaxed parent, an awake and enthusiastic child, a mild anxiolytic, although probably with little pharmacological effect and EMLA cream. The results are not perfect, but generally lead to an enjoyable day for the anaesthetist and theatre staff and hopefully an undisturbed child and parents.

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Failed obstetric intubation

The report by Dr McClune *et al.* (*Anaesthesia* 1990; 45: 227-8) was interesting and, as a regular user of the laryngeal mask, I hoped that this would provide the answer to a difficult problem. However, this did not prove to be the case.

A 22-year-old primiparous patient, who weighed 105 kg, presented at term with a spontaneous rupture of membranes and a high head. She produced thick, fresh meconium and it was decided to proceed to emergency Caesarean section. She was given intravenous ranitidine 50 mg and sodium citrate 30 ml orally, in the anaesthetic room, and thiopentone 375 mg and suxamethonium 100 mg, after pre-oxygenation, with cricoid pressure applied. The tip of the epiglottis, a Grade III laryngoscopy, only was visible. The trachea was not intubated. A further attempt using a 7.0-mm tube threaded over a gum elastic bougie also failed. The patient's oxygen saturation decreased to 60% and oxygenation was carried out using a facemask and airway, with the cricoid pressure maintained. The saturation rose to 90% but as the airway was very difficult to maintain I decided to use the laryngeal mask. A size 4 laryngeal mask was placed without difficulty but ventilation of her lungs was impossible even after release of cricoid pressure. The oxygen saturation again decreased to 64% and the laryngeal mask was removed. Ventilation was continued through a bag and facemask until she began to breathe spontaneously. She was then turned onto her left side and spinal anaesthesia instituted. LSCS was performed and a live female infant delivered with Apgar scores of 9 at 1 minute and 9 at 10 minutes.

The laryngeal mask should be an essential part of the

obstetric theatre's equipment but it will not always be the answer to the anaesthetist's prayer.

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A reply

We agree with Dr Christian, that the laryngeal mask is not a fail-safe device in the case of unsuccessful tracheal intubation for emergency Caesarean section. The same applies to any piece of equipment reserved for securing the airway after unsuccessful intubation, such as the gum elastic bougie. It is well recognised that the laryngeal orifice can be occluded by the epiglottis during insertion of the laryngeal mask.¹

Dr Christian was fortunate that adequate ventilation of the patient was possible using a facemask and rebreathing bag. Manual ventilation via a facemask was not effective in the case we described.

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Reference

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Laryngeal mask and magnetic resonance—a caution

The letter from Dr C. Rafferty and colleagues (*Anaesthesia*, 1990; 45: 590-1) about the laryngeal mask and magnetic resonance imaging was interesting. It is certainly a great benefit to be able to use a device that guarantees a clear airway, and avoids the necessity for tracheal intubation. However, their bold assertion that the laryngeal mask airway is suitable for magnetic resonance imaging because it '... is made of plastic and therefore has no ferromagnetic properties', is an over-simplification. It is indeed sufficient to be certain that any devices within the region to be studied are free of magnetic properties when performing simple magnetic resonance imaging; however, if magnetic resonance spectroscopy is to be performed it is also necessary to ensure that the device has no magnetic

resonance at the frequency of interest which could be mistakenly attributed to the tissue under study. We have found while performing ¹³C spectroscopy that several varieties of plastic tubing, particularly those containing silicone, have resonances that are inseparable from the tissue under investigation. It must be remembered that although a device may be altogether suitable for magnetic resonance imaging, it is not necessarily suitable for spectroscopy.

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Successful use of propofol in status epilepticus

Propofol is not a recognised drug treatment for epilepsy and its use in epileptic patients remains controversial. Some reports^{1,2} suggest it is epileptogenic and does not possess anticonvulsant activity.³ However, several clinical trials demonstrated reduced seizure duration after electroconvulsive therapy using propofol.⁴⁻⁶ Two other published cases cite successful treatment of status epilepticus with propofol,^{7,8} but lung ventilation was required. A recent review article was unable to make a firmer conclusion than 'propofol appears to possess anticonvulsant properties clinically'.⁹

We wish to report a case where propofol was used

successfully without ventilatory support to treat status epilepticus, after recognised treatments had failed.

Anaesthetic staff were asked to see a 65-year-old 70-kg woman, admitted 4 days previously in status epilepticus, to 'paralyse and ventilate' her. A left-sided meningioma was excised in 1967 with no recurrence but with sequelae of mild right hemiparesis, dysphasia and post-craniotomy epilepsy. Her usual treatment consisted of phenytoin 300 mg and vigabatrin 2 g daily.

She suffered continuous clonic movements of her right arm, shoulder and leg with aphasia in the 4 days since admission. She maintained adequate ventilation but had

limited awareness of her surroundings. Treatment included parenteral phenytoin 400 mg/day, phenobarbitone 240 mg/day, chlormethiazole 0.8% infusion up to 10 ml/minute, diazepam boluses up to 20 mg/hour, and a single intramuscular injection of paraldehyde 5 ml, all without effect.

Treatment with thiopentone 250 mg in increments stopped the convulsions for 5 minutes but at the cost of loss of consciousness and voluntary airway control. This was considered to be unsatisfactory and prompted a trial of propofol one hour later.

A 30-mg bolus of propofol abolished the fits, and infusion of 175 mg/hour (2.5 (mg/kg)/hour) with continuation of phenytoin 300 mg/day and phenobarbitone 200 mg/day gave excellent seizure control with only occasional isolated twitches. The patient was drowsy but maintained her own airway and satisfactory breathing as monitored by clinical observations, pulse oximetry and arterial blood gas analysis. Attempts at reducing the rate of infusion resulted in further fits until 3 days later when weaning was successful.

She suffered no further seizures and slowly recovered to her former state.

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Dilution of Diprivan

We read with interest the letter by Drs Wright and Filshie (*Anaesthesia* 1990; **45**: 421), who note that the quality of induction when using Diprivan in children is much improved if diluted with saline before injection. We were uncertain from the letter whether dilution took place immediately before administration in the cannula, or whether the authors recommend premixing of Diprivan with saline. Diprivan may be administered via a Y-piece close to the injection site into infusions of sodium chloride 0.9%. However, premixing in a bag of saline would lead to breakdown of the emulsion, and ICI do not recommend this. If diluted Diprivan is required, Diprivan may be

diluted with 5% dextrose (Intravenous Infusion BP only) in PVC infusion bags or glass infusion bottles. Dilutions, which must not exceed 1 in 5 (2 mg propofol/ml), should be prepared immediately before administration and used within 6 hours of preparation.

We also remind anaesthetists that Diprivan is not licensed for paediatric use in the UK.

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K.J. HOPKINS
R. ALBANESE

Soft tissues and difficult intubation

We were interested to read the correspondence by Wilson and John¹ about the Mallampati test to predict difficult tracheal intubation.² Reference was made to early correspondence by ourselves about unreasonably high rates of false positive prediction of difficulty.³ One of the main points we made at that time was that the test was 'subjective and far too inconsistent in any given patient'. This seemed obvious with single observer assessment so we did not go on to perform interobserver comparisons. Inconsistency must surely be seen as inevitable when both the tongue and soft palate are extremely mobile structures for a 'wavering, ill defined boundary'.

It was interesting also to note that Wilson and John were able to obtain a definition of how to differentiate between the class III and class IV assessment used by Young and Samsoon.⁴ This specific question was also directed at them in our correspondence but no answer was forthcoming.

Soft tissue factors in difficult intubation have undoubtedly not received adequate attention because of the difficulty of making objective assessments of tongue size. X ray laryngoscopy studies have indicated the importance

of control of the hyoid bone with the tip of the laryngoscope blade and the mechanisms which may prevent this.⁵ These studies also make it quite clear that the 'anterior larynx' is a misconception. The problem is that the blade tip fails to negotiate the tongue to reach the hyoid and so remains in a relatively posterior position to that normally achieved. This posterior placement is responsible for the impression that the larynx is further forward than normal.

The most important point about the Mallampati test concerns the whole question of whether or not it is even likely that it reveals relative tongue size. An abnormal test would appear to have at least the same chance of being caused by a relatively long soft palate as any enlargement of the tongue. This matter seems to be repeatedly overlooked by advocates of the test. It seems a reasonable clinical preception that many overweight sleep apnoea patients have relatively large tongues and soft palates, but we know of no evidence to suggest that these two features inevitably occur simultaneously.

Finally, we feel that we should draw attention to our

paper in which modern radiological perceptions of movement at the upper part of the cervical spine are reviewed.⁶ It is appropriate to describe movements at the occipito-axial joint rather than at the atlanto-occipital joint as do Wilson and John.

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'Overdose' of vecuronium

Histamine release after vecuronium administration with resultant cardiovascular collapse, is again reported.¹ There are also a number of reported cases of bradycardia and asystole after bolus administration of the recommended dose of the drug. These have occurred with a combination of opioids, notably fentanyl, and potent vagal stimuli,^{2,3} and appear to contradict the emphasis the manufacturers of the drug have placed on its lack of cardiovascular side effects. We report an accidental overdose of vecuronium given to a haemodynamically unstable child with no adverse effects.

A 9.7-kg child, aged 23 months, with Down's syndrome had corrective cardiac surgery for an atrioventricular canal defect. The postoperative course was complicated by a poor cardiac output state, intermittent junctional tachycardias, bronchopneumonia and a tension pneumothorax which resulted in a cardiac arrest. The child still required high concentrations of inspired oxygen and controlled ventilation of the lungs 12 days later. Cardiovascular support from dobutamine (12.5 (μ g/kg)/minute, aminophylline (0.8 (mg/kg)/hour), and prostacyclin (20 (nanog/kg)/minute) was also required. A morphine infusion (20 (μ g/kg)/hour) with occasional bolus administration of diazepam was used for sedation. Paralysis was maintained throughout with a vecuronium infusion (0.15 (mg/kg)/hour). The child's liver function was abnormal and the urine output was frusemide dependent, although plasma creatinine was within normal limits. Vancomycin and ceftazidime were given for bronchopneumonia. An incorrect rate was set after a change of infusion pump, which delivered 37 mg (3.83 (mg/kg)/hour) of vecuronium intravenously over one hour. All haemodynamic variables remained virtually unchanged during the infusion and over the next 24 hours. The degree of cardiovascular support was not altered during this time. The infusion was restarted again 18 hours later when significant diaphragmatic movement was seen after tracheal suction. There was marked tetanic fade and a train-of-four count of two, but only after a tetanic

stimulus. Plasma samples taken at the time were unfortunately found to be unsuitable for drug assay. The child died 5 days later from overwhelming sepsis.

The dose of drug given represents a 50-fold overdose of the manufacturer's recommended infusion rate (0.5-0.8 (mg/kg)/hour).

A literature search failed to find any previous reports of such a large overdose of vecuronium; however, a similarly large overdose of atracurium given by infusion was reported.⁴ This also resulted in no significant haemodynamic effects although the infant was noted to have marked skin flushing. This is in contrast to a report⁵ after a 10 times bolus overdose of atracurium in a small infant which resulted in cardiovascular collapse and bronchospasm.

This case highlights the high therapeutic index afforded by vecuronium particularly when given by infusion. No clinical evidence of histamine release was seen in this child, contrary to the two reports of atracurium overdose. Vecuronium must remain one of the most cardiovascularly stable of the presently available neuromuscular blocking drugs, except in an extremely small number of susceptible patients.

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Increased use of gum-elastic bougie in clinical practice

There is evidence further to our recent paper¹ to suggest that there is increased use of the gum-elastic bougie in cases of difficult tracheal intubation. However, the bougie may not always be used in an optimal manner.

A survey of methods chosen by British anaesthetists to facilitate tracheal intubation was conducted at a Symposium on Difficult Intubation in 1982. This showed

that the respondents were evenly divided between those who preferred a stylet to a bougie as a first choice in cases of difficult intubation.²

A questionnaire was used to assess which simple methods (gum-elastic bougie, stylet or other) anaesthetists in Cardiff would use first in a case of difficult intubation in 1988. It also asked whether the laryngoscope was left in the

Table 1. Answers to questionnaire (Cardiff 1988) (*n* = 51).

First choice method	<i>n</i>	%
Gum-elastic bougie	46	90
Stylet	4	8
Other	1	2
Use of laryngoscope during intubation		
Yes	40	88
No	11	22
Position of tube		
0°	36	70
−90°	10	20
Other	5	10

mouth and if the tube was rotated during difficult intubation manoeuvres with a bougie.

There were 51 replies to the questionnaire; this is a response rate of 87%. A great majority of anaesthetists

(90%) would use a gum-elastic bougie and most (88%) left the laryngoscope in the mouth while the tube was passed (Table 1). However, only 20% used the −90° tube position over the bougie; five (10%) respondents volunteered that they rotated the tube continuously through 360° while passing it over the bougie. The tube positions have previously been described.¹

We hope that in addition to leaving the laryngoscope in the mouth the number of clinicians using the −90° tube position over a bougie as a first choice will increase since it has been shown that this offers the best possible chance of success in a case of difficult intubation.¹

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S. DOGRA
R. FALCONER
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Unexpected airway obstruction

An 80-year-old woman presented for surgery with an acutely ischaemic left leg. She was in atrial fibrillation at a rate of 80 beats per minute but no other abnormal physical findings were noted. She was scheduled for an urgent exploration of left popliteal artery.

She underwent an uneventful but prolonged (3-hour) anaesthetic for exploration of the left popliteal artery. Tracheal intubation was atraumatic with easy passage of an 8.0-mm cuffed PVC tracheal tube. Her trachea was extubated and she was transferred to the recovery room at 1700 hours. At 2000 hours she was returned to theatre for exploration of the wound because of haemorrhage.

Anaesthesia was induced in the theatre after pre-oxygenation, using a standard rapid sequence introduction: intravenous etomidate 10 mg and suxamethonium 75 mg were given. Intubation was attempted with a 8.0-mm cuffed tracheal tube, but as this would not pass easily a 7.0-mm tracheal tube which was cut to 20 cm was used and passed easily.

A capnograph indicated carbon dioxide in the expired gas. Hand ventilation, however, proved very difficult. Both sides of the chest moved equally but not very much. Breath sounds were equal with no wheeze or evidence of a pneumothorax. It was impossible to ventilate her adequately using a Manley Pulmovent with safety pressure set at 7.0 kPa.

A laryngoscope was inserted and the tracheal tube removed. A view of the subglottic area at this time showed an oedematous, narrowed trachea. It was decided to replace the tracheal tube with the same size but longer (23 cm) tube. This was passed through the cords and narrowing and the cuff inflated. Manual ventilation was now easy. Airway pressures with automatic ventilation were 2.0 kPa with equal air entry.

Her breathing was normal after extubation with no stridor or other evidence of airway obstruction. She was admitted to the ITU for observation overnight. Intravenous hydrocortisone 200 mg and humidified oxygen-enriched air was given. Subsequently she made an uneventful recovery.

The case illustrates some important points. The possibility of trauma to the larynx and subglottic region with associated swelling in the upper airway should be remembered in patients recently intubated. A range of smaller, as well as longer, tracheal tubes should be readily available. Once again it emphasises that if there is any doubt as to the correct placement of a tracheal tube, it should be promptly removed.

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A.I. McEWAN
J.N. CASHMAN

Central venous cannulation: two complications

A 63-year-old man with multiple organ failure was referred to our intensive therapy unit. The transfer, by the Shock Team, was uneventful and the patient arrived stable and comfortable. Shortly after he was settled in bed, a nurse found the side-arm from the introducer sheath of his pulmonary artery catheter lying loose in the bed clothes. The hole in the hub was covered with a finger and the patient tilted head-down. The sheath and catheter were then removed together. The gap between the sheath and the pulmonary artery catheter was blocked with clot (Fig. 1). This had prevented either back-bleeding or air entrainment during the many recent changes in his position.

It is not certain when the side-arm became detached from its hub but the most likely times are when the patient

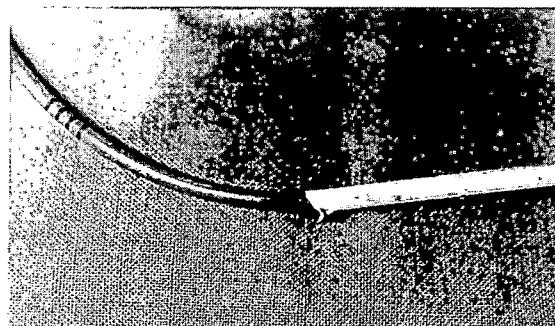


Fig. 1.

was lifted to and from the transfer trolley whilst being moved about his bed. If he had been dragged (rather than lifted) considerable force could have been applied to the side-arm. There is no relevant British Standard for introducer sheaths, but the Vygon product (#1148.08) appears well made and may well have been subjected to unreasonable demands.

A large cannula that opens the superior vena cava to air can kill: this man was very lucky to have a second 'complication' that protected him from harm.

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D.L. PAUL

Provoked gastro-oesophageal reflux?

Bogod *et al.* (*Anaesthesia* 1990; **45**: 279–84) have detected awareness during general anaesthesia for both elective and emergency Caesarean section. They recorded lower oesophageal contractions provoked by the infiltration of a balloon in the lower oesophagus.

The inflation of a balloon in the lower oesophagus also provokes a relaxation of the lower oesophageal sphincter where its sphincter pressure decreases to equal gastric pressure.¹ This reflex relaxation normally occurs in response to gastric distension, lasts for 5–30 seconds and is the predominant mechanism that causes gastro-oesophageal reflux in conscious humans.² Gastro-oesophageal reflux does not always follow when the lower oesophageal sphincter has relaxed, this depends on the volume of gastric contents, gastric pressure and the position of the patient.^{1,2} However, during anaesthesia, these reflex relaxations of the lower oesophageal sphincter are inhibited,³ but may still occur if the patient was aware and therefore conscious.

Did regurgitation of gastric contents occur in the study patients? Provoked gastro-oesophageal reflux with this equipment should be investigated further if it is to be used routinely in these patients at high risk of acid aspiration pneumonitis.

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A reply

Thank you for the opportunity to reply to this letter. There is a theoretical risk of gastric reflux associated with the use of the oesophageal probe. However, this is not a problem in practice, and regurgitation was not seen in any of our patients. Despite this, we do not advocate the use of this device in the presence of an unprotected airway in patients at risk from acid aspiration, and recommend that, as in our study, the probe be passed only after the airway is secured by a correctly placed tracheal tube with cuff inflated. The probe should be removed before tracheal extubation.

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D.G. BOGOD

Malfunction of Servo ventilator in manual mode

We wish to report a serious malfunction that occurred in the manual ventilation accessory of the Servo 900 D ventilator (Fig. 1). The control lever A (see Fig. 2(a) and (b)) is moved through 90° in the change from manual to automatic ventilation. This control lever pivots, lowering the rod F, the mushroom valve E and magnet H, and seals the manual breathing bag (at D) from the fresh gas flow. A small grub screw (Fig. 3) connecting rod F and lever A worked loose, so that on return of the lever from the upright to the horizontal position incomplete travel of the rod resulted, and incomplete descent of the mushroom valve and magnet so that the manual bag was not completely sealed off. If there is a Heidbrink valve with the manual ventilation bag and it is open, then no ventilation occurs since no positive pressure develops. If the Heidbrink valve is closed the patient would be ventilated in parallel with the manual bag after the bag fills and reaches a pressure. The amount of ventilation depends on the relative compliance of the patient and bag. The expired minute volume meter reflects expiration from both the patient and bag and may indicate adequate ventilation, although the patient's lungs are really hypoventilated. The manual ventilation bag will also inflate in time with the ventilator which it should not do in the automatic ventilation mode.

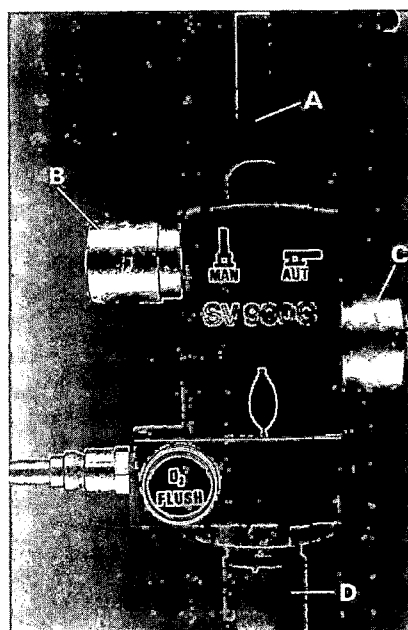


Fig. 1.

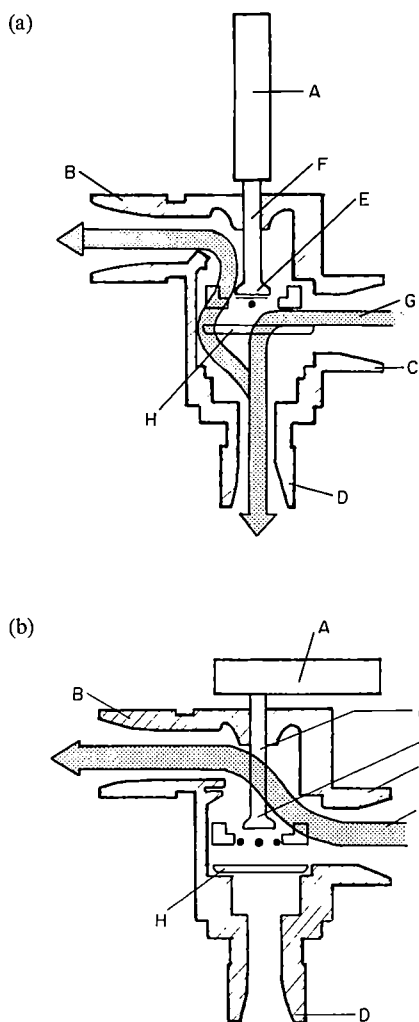


Fig. 2(a) and (b). A, control lever; B, patient inspiratory hose attachment; C, ventilator attachment; D, attachment for manual breathing bag; E, mushroom valve; F, rod; G, direction of gas flow; H, magnet. The arrow indicates the grub screw.

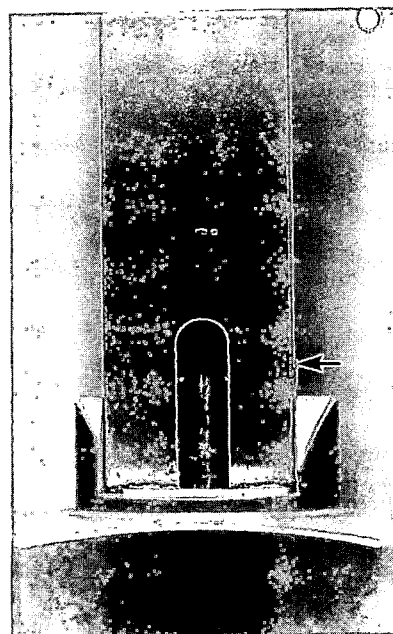


Fig. 3.

This malfunction occurred on completion of a lobectomy, when one lung anaesthesia was used when, after re-expansion of the lung using the manual accessory, and on resumption of two-lung ventilation; it was noticed that the lung was not expanding and the expired minute volume alarm was activated. This demonstrates the importance of clinical monitoring: observation of lung expansion and the use of alarms. It is useful if the alarms are reset immediately rather than allowed to be mute for 2 minutes.

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J.J. OWENS
J. KEOGH

Editor's note. We have received no reply from the manufacturers.

Interlocking connexion in anaesthetic systems

Disconnexion of anaesthetic tubing remains a potential cause of mortality and serious morbidity during anaesthesia despite the use of ventilator alarms. This is particularly the case for ENT, dental, ophthalmic and neurosurgery.

The Figure shows a design for an interlocking system which would dramatically reduce the possibility of disconnexion while, at the same time, allow quick and easy disengagement when required.

The drawing is of the prototype (patented) made in steel and is therefore a rather crude representation. However, it can be seen that the modifications are external to the tube and therefore have no adverse effect on internal diameter or gas flow. There are no internal parts to the tube which could break off during use.

We consider that a similar principle could be applied to other connexions throughout the anaesthetic system.

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Eire

J.F. WALSH
P.V. WANI

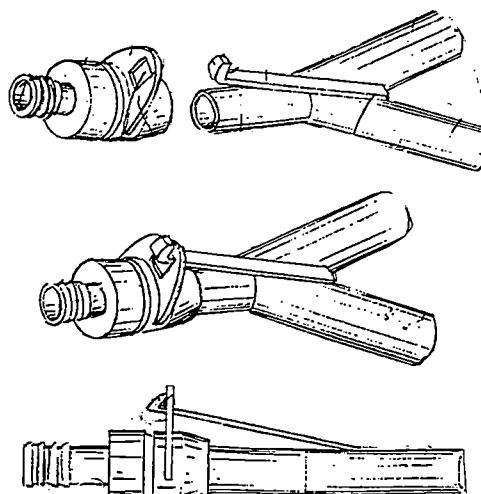


Fig. 1. Three views of a quick-release connector for breathing systems.

Fixation of the laryngeal mask airway

There is a small problem when the laryngeal mask airway (LMA) is used. A satisfactory method to secure position of the LMA is required and the 'Tracheal Tube Holder' (TTH) manufactured by Portex, Hythe, seems ideal.

The device supports the LMA firmly over a length of some 8 cm and prevents it from movement or kinking under the weight of the anaesthetic tubing. The bite block prevents compression of the tube between the teeth during recovery and facilitates removal at this stage. Use of a bite block is recommended by the manufacturer of the LMA.¹

This combination of fixation and bite block represents an advantage over the fibroscope protector described by Dr Marks (*Anaesthesia* 1990; **45**: 259). The TTH is easily inserted after the LMA is satisfactorily positioned. It is

placed in the opposite side of the mouth from the LMA and is held open while the two are slid together. The two can then be slipped apart during recovery and the TTH kept in place while the LMA is removed.

The device costs less than £5 and is re-usable after cleaning.

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Reference

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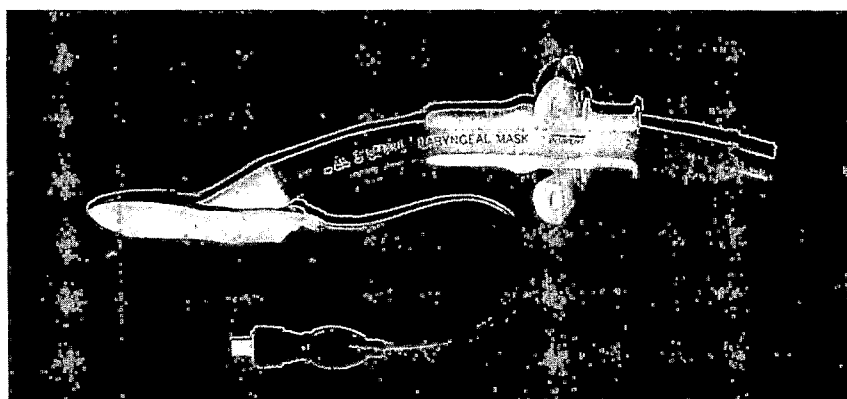


Fig. 1.

Attachments to double lumen bronchial tubes

We would like to report a novel arrangement of ventilator tubing for attachment to a double lumen bronchial tube in order to prevent soiling from one lumen to another and to avoid an increase in the deadspace of the system.

A woman in her 40s with past history of arteriovenous malformations (AVMs) affecting both kidneys was referred to the cardiothoracic service in respiratory failure after a massive haemoptysis. Chest X ray showed unilateral lesions

on the right side which were presumed to be further AVMs in the lung. It was decided to isolate the affected bronchus to prevent further soiling of the relatively healthy left lung, so a double lumen bronchial tube was passed. Unfortunately, in view of continued massive bleeding from the right lung, the left bronchial system was still at risk from contamination by blood which reached as far proximally as the catheter mount. An extra set of ventilator



Fig. 1. The layout of ventilator tubing is shown with the inspiratory system in black, and the expiratory system in white.

tubing was connected to and from each lumen of the catheter mount. A Y-piece was used to bifurcate the inspiratory limb and take inspiratory gases to each of the lumina. Expiratory gases were returned from each lumen to converge on a Y-piece at the expiratory limb in a similar manner.

Thus both inspiratory and expiratory gases were split proximal to the catheter mount in a simple fashion without any increase in the deadspace of the system. The volume of

a standard length of ventilator tubing is approximately 500 ml, so this prevents cross contamination resulting from all but the most profuse haemorrhage, and the need for the use of two synchronised ventilators was avoided.

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Preparation and use of propofol

We are grateful to the Editor for the opportunity to draw the attention of readers to the fact that a small number of patients have recently been reported to have developed a postoperative pyrexia and infections after anaesthesia with propofol (Diprivan). Many of the cases have been detailed in a report recently published by the Center for Diseases Control (CDC) in the United States.¹

The CDC reviewed the working practices of the anaesthetic staff concerned after reports of clusters of postoperative pyrexia or infection in patients from four US hospitals after anaesthesia involving Diprivan. The report concludes that aseptic conditions were not achieved during administration of Diprivan. Unopened ampoules of Diprivan from the same lots in use at the hospitals were analysed by CDC and shown to be intrinsically sterile and free of contamination with endotoxins.

It is important to keep in mind the Diprivan is a soyabean oil emulsion. Aseptic techniques must always be maintained during handling since Diprivan contains no antimicrobial preservatives and the vehicle is a potential

growth medium for microorganisms. Diprivan should be drawn into a sterile syringe immediately after the ampoule is opened and administration should start promptly. Each unit of Diprivan is intended for use in a single patient and the syringe and any unused portion must be discarded at the end of the surgical procedure.

Failure to apply aseptic handling procedures, as with the intravenous administration of any drug, may result in microbial contamination which may cause fever and (or) other adverse consequences for the patient.

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Cheshire SK10 4TF

J.S. PATTERSON
K.J. HOPKINS
R. ALBANESE

Reference

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Erratum

Anaesthesia, 1990, Volume 45, pages 683-684

Bradypnoea during patient-controlled analgesia

Through an oversight, Dr A.J. Matthews' name was omitted from the list of authors of the above letter, for which we apologise.

Book reviews

Pharmacology of neuromuscular function 1003
W.C. BOWMAN

Clinical aspects of O₂ transport and tissue oxygenation 1003
K. REINHART AND K. EYRICH

Drugs in anaesthesia and intensive care 1003
M.P. SASADA AND S.P. SMITH

Books received 1004

Pharmacology of neuromuscular function

W.C. BOWMAN. Pp. x+316. Wright, 1990. £45.

It is misleading to judge from the subtitle of this book that it is a second edition update of the excellent concise review which has been a bench book in anaesthetic departments for the last 10 years. This is a new book, and a comprehensive monograph on the state of knowledge of neuromuscular physiology and pharmacology. It is written by a pure scientist, and as such confines itself to the explanation of clinical and experimental phenomena by the clear use and interpretation of the current evidence. The book is therefore free of clinical opinion and allows the anaesthetic readers to concentrate on the scientific basis of their clinical practice. There are many new illustrations, both experimental results, and clear descriptive drawings. The 51 pages of references grouped at the end of the book, presumably to save considerable repetition, would be enough on their own to make the book invaluable to anyone embarking in research in this field.

The reason why this book should find its way into every anaesthetic department is much more simple: it is because the understanding of neuromuscular pharmacology is so important in any teaching programme in anaesthesia that those who have read this new edition are already busy revising their lectures and tutorials. There is, for example, the first clear explanation of the pharmacological basis of fade of the train-of-four, a rather fundamental component of daily clinical monitoring. In addition, the full range of drugs past, present and future which relate to neuromuscular transmission are reviewed. The price reflects both the more substantial nature of the book, and also a great improvement in the presentation.

D.E.F. NEWTON

Clinical aspects of O₂ transport and tissue oxygenation

Edited by K. REINHART AND K. EYRICH. Pp. xiii+511. Springer Verlag, 1989. DM 158.

The title of this book provides a reasonably clear indication of its contents. There are five main sections; each of these has 4–12 papers from authors in England, USA, Canada and six countries in Europe, and provides brief review articles on many 'aspects' with considerable variety of approach. This is not a book to read from beginning to end (as your reviewer was obliged to do) but selective reading would offer much of interest, albeit usually with a necessary brevity.

The initial section on Physiology deals with many of the processes involved in the transfer of oxygen from the lungs to the tissues in health and sickness and includes descriptions of supply dependence, hypoxia in multiple organ failure and reperfusion injury. The next section on Monitoring considers the significance of pre-operative assessment, peri-operative myocardial ischaemia, as indi-

cators of tissue oxygenation and the use of oximetry. The third section, Therapy, contains papers on the variety of methods of ventilatory assistance, extracorporeal and hyperbaric gas exchange and pharmacological manipulation of cardiac and peripheral circulatory function. The shortest section, Anaesthesia, relates effects of anaesthesia on respiratory function and on myocardial and hepatic supply of oxygen and is followed by a section, Cardiopulmonary Resuscitation and Cardiac Transplantation, on cardiac arrest and peripheral circulatory, including cerebrovascular, failure and cardiorespiratory function after cardiac transplantation.

Anaesthetists and those involved in intensive care will find most of the book useful but particularly those chapters which focus attention on the relationship between peripheral circulatory function, tissue oxygenation, metabolism and the potential for therapeutic intervention; in these areas the book has a cohesion that is lacking elsewhere which seems inevitable with this type of multi-authored collection. The chapters are mostly well referenced to provide 'ways into' the literature.

The publishers claim that this is a monograph; if this is so then the term is without meaning applied to a text that has such a diversity of sources and subject matter. It would not be useful, and indeed would probably confuse the relatively inexperienced, who should not be encouraged in its direction; however, I enjoyed my task and recommend it to the practitioner who can gain access to a copy.

D. WEATHERILL

Drugs in anaesthesia and intensive care

M.P. SASADA AND S.P. SMITH. Pp. 266. Castle House, 1990. £24.

The scope of this compact little book is clearly defined by the authors: it is to encompass a summary of the drugs that anaesthetists may encounter and in a standard format for the presentation of this information. The most striking feature of the book is the abandonment of chapters and 'artificial categorisation' of drugs. The 172 drugs described are listed in alphabetical order with from one to two pages devoted to each agent. There are neither illustrations, references nor an index.

The standard format used to describe all of the drugs consists of 15 headings which cover all pharmacodynamic and pharmacokinetic aspects of the agent. This rigid structure certainly speeds the extraction of information. Most of us can remember only too well the need to search through large tomes in search of a small specific point, always wondering if it was hidden in a distant unrelated chapter. It takes only a matter of seconds to locate the section required on a particular drug in this book. Therein lie its strengths and weaknesses. It is an excellent *aide memoire* but because of its limited size and scope it will always be an adjunct to the larger standard textbook which gives the

background principles of pharmacology and all other details.

The drugs covered include almost all anaesthetic-related agents and those drugs used for medical conditions which are commonly encountered by anaesthetists. Some might question the omission of gallamine and I would take issue with the emphasis given to certain agents. Does diethyl ether really merit more attention than thiopentone, propofol, enflurane or isoflurane? The factors covered in the standard format used to describe the drugs are excellent although perhaps an approximate indication of a drug's cost would be useful and also the inclusion of North American names where they differ from those used in this country.

For whom is this book? The trainee anaesthetist will certainly find this a useful source for revision, especially before the part II exam. However, the clarity of layout achieved by a description of each drug in isolation and purely in alphabetical order does make comparison between the various members of a group of drugs more difficult. Furthermore, the examination candidate will not benefit from the sprinkling of silly errors throughout the text. Nonetheless, any candidate who can learn to organise his or her written or oral answers in a standard format, as used here, should do well. Beyond its role as an examination aid this book should prove useful to all grades of anaesthetists, both for browsing and for use in urgent situations where a rapid clear answer is sought.

This book fills an important gap between the major anaesthetic pharmacology textbooks on the one hand and the British National Formulary and drug data sheets on the other; and I recommend it.

C.C. CALLANDER

Books received

We thank the publishers for the following books, some of which may be reviewed in future issues of *Anaesthesia*.

Anesthesia, 3rd edn (2 volumes)

Edited by R.D. MILLER. Pp. 2418. Churchill Livingstone, 1990. £115.

Clinical neuroanesthesia

Edited by R.F. CUCCHIARA AND R.D. MICHENFELDER. Pp. xi + 576. Churchill Livingstone, 1990. £57.50.

Clinical oxygen pressure measurement II

Edited by A.M. EHRLY, W. FLECKENSTEIN, J. HAUSS AND R. HUCH. Pp. 436. Blackwell Scientific, 1990. £110.

Handbook of critical care, 3rd edn.

Edited by J.L. BERK AND J.E. SAMPLINER. Pp. xix + 812. Churchill Livingstone, 1990. £32.50.

Réanimation et médecine d'urgence

G. FRANÇOIS, P. CARLI, P. BOULETREAU, C. GRANTHIL *et al.* Pp. x + 325. Masson, 1990. 119F.

Major chemical disasters—medical aspects of management

Edited by V. MURRAY. Pp. 204. Royal Society of Medicine, 1990. £15.

Patient-controlled analgesia

Edited by F. MICHAEL FERRANTE, G. W. OSTHEIMER AND B.G. COVINO. Pp. xi + 244. Blackwell Scientific, 1990. £32.50.

Safety Action Bulletin

The attention of readers is drawn to the notice below which was promulgated by the Department of Health, the Scottish Home and Health Department and the Welsh Office and the Department of Health and Social Services for Northern Ireland.

Reminder to report incidents in accordance with HC(88)51 (and equivalent in other parts of the UK)

A number of recent incidents have caused concern that the guidance in HC(88)51 (and other parts of the UK's equivalents) is not being followed.

The recent serious incidents highlight the need to remind operatives that the guidance in HC(88)51 (and other parts of the UK's equivalents) should be followed explicitly.

In one of these incidents the death of a patient was related to the equipment being used but no report was made to the Procurement Directorate (PD) until 10 days afterwards. This event may have had serious implications for other users but the time lapse prevented immediate investigation by PD and consequently led to a delay in deciding whether advice needed to be issued to the NHS.

The Department is concerned that such delays could have serious implications for patients or staff.

It is imperative, therefore, to report such incidents promptly and also to ensure that related equipment is isolated until investigated by the Department's representatives, in accordance with the guidance.

Management should ensure that all staff are aware of their responsibilities and the correct procedure for reporting incidents and defects.

The following are the four names and addresses currently appropriate for the four countries of the United Kingdom.

England: Mr J.P. Nash, NHS Procurement Directorate, Room 422, 14 Russell Square, London WC1B 5EP (Tel: 071-636 6811 Ext 3328, Fax: 071-637 8990).

Scotland: Miss K. Glancy, SHHD, Room 54H, St Andrew's House, Edinburgh EH1 3DE (Tel: 031-422 2428).

Wales: Mr G.A. Willis, Welsh Office, Health Management System, Personnel Division, Cathay's Park, Cardiff CF1 3NQ (Tel: 0222 823641).

Northern Ireland: Mr O. Boyle, Estate Services Division, Stoney Road, Dundonald, Belfast BT16 0US (Tel: 02318 3299 Ext 2425).

Notice to contributors to *Anaesthesia*

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Contributors are requested to submit two copies of manuscripts. They are also advised to retain a third copy as the Editors cannot accept responsibility for the loss of manuscripts in the post. The covering letter should be signed personally by all the authors and careful consideration should be given to the decision to include more than five authors. Articles should be forwarded to:

Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK.

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Articles for *Anaesthesia* should be prepared in accordance with *Uniform requirements for manuscripts submitted to biomedical journals* (British Medical Journal 1979; 1: 532-5) except that the titles of journals in the reference section should be given in full (see below). A reprint of these requirements of which this notice is a summary, can be obtained from the *British Medical Journal* price 50 pence (UK).

Type manuscripts on white bond paper, 20.3 × 26.7 cm or 21.6 × 27.9 cm (8 × 10½ in. or 8½ × 11 in.) or ISO A4 (212 × 297 mm) with margins of at least 2.5 cm (1 in.). Use double, and preferably triple, spacing throughout, including the references. Please do not use a dot matrix printer, particularly one with poor quality descenders or ascenders. Unseparated, fan-folded manuscripts may be returned to the author. The manuscript should consist of the following sections in this order each beginning on a new page: title page, summary and key words, text, acknowledgements, references, individual tables, and legends for figures.

Number pages consecutively, beginning with the title page.

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These should be typed at the left-hand side of the page above the paragraph which they precede.

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Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables and legends by arabic numerals. References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration. Use double or treble spaced typing.

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Avoid using abstracts as references except those published in a recognised journal. 'Unpublished observations' and 'personal communications' may not be used as references, although references to written, not verbal, communications may be inserted (in parentheses) in the text. Include among the references manuscripts accepted but not yet published; designate the journal followed by (in press) in parentheses. Information from manuscripts submitted but not yet accepted should be cited in the text as (unpublished observations) in parentheses.

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(continued overleaf)

Examples of correct form of references
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JOURNAL

Standard journal article—(List all authors)

SOTER NA, WASSERMAN SI, AUSTEN KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *New England Journal of Medicine* 1976; **294**: 687–90.

Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

WEINSTEIN L, SWARTZ MN. Pathogenic properties of invading micro-organisms. In: SODEMAN WA Jr, SODEMAN WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*. No. 69) [DHEW publication No. (HSM) 72-1036].

OTHER ARTICLES

Newspaper article

SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

Magazine article

ROUCHE B. Annals of medicine: the Santa Claus culture. *The New Yorker* 1971 Sept 4: 66–81.

TABLES

Do not include tables in the text. Start a new sheet for each table and space the material adequately. The author(s) name(s) should appear in the top right-hand corner.

Indicate the approximate position of each table in relation to the subject matter of the text in the left-hand margin of the appropriate page on the manuscript. Do not submit tables as photographs. Number tables consecutively with arabic numerals. Supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes. Explain in footnotes all non-standard abbreviations that are used in each table. For footnotes, use the following symbols in this sequence: *, †, ‡, §, ||, ¶, **, ††, etc. Identify statistical measures of variations such as SD and SEM. *Legends for tables* should appear on the face of the table.

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Statistics and measurements should be given in figures except that numerals one to nine should be in words if not followed by a measurement symbol (e.g. 'two patients' but 2.0 mg). The *Système International* (SI) will usually be used except that vascular pressures will be recorded in mmHg and cmH₂O. Imperial measurements will not be used except in an historic context. The 24 hour clock will be used.

Contributors are advised to study the *SI Conversion Tables* provided in the January 1978 issue of *Anaesthesia, Units, Symbols and Abbreviations* (*A Guide for Biological and Medical Editors and Authors*) published by the Royal Society of Medicine, London W1M 8AE, and *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1**: 532–5).

The statistical tests used in the report should be stated clearly. Results should include 95% confidence limits for the main findings and probability estimates.

LETTERS FOR PUBLICATION

Should be addressed to Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK.

Letters should be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters above. The degrees and diplomas of each author must be given in a covering letter which must be signed personally by all the authors.

Correspondence presented in any other style or format may be subject to considerable delay and may be returned to the author for revision.

The editors regret that failure to comply with the above requirements may result in a delay in publication of accepted papers.

REVIEW JOURNALS

This journal is covered by *Current Contents*, *ASCA*, the *Science Citation Index* and *Index Medicus*.

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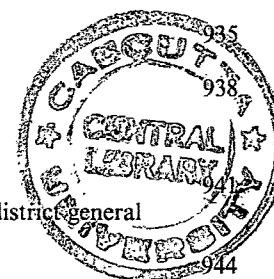
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Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 45 Number 12 December 1990




The Association of Anaesthetists of Great Britain and Ireland
Patron: HRH The Princess Margaret, Countess of Snowdon



Published for the Association by
Academic Press Grune & Stratton
London San Diego New York Boston
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ISSN 0003-2409



Ghost bat.
Macroderma gigas.
Highly sensitive ears.
Brain discriminates and
interprets complex
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Is this an anaesthetist's role model?

*Anaesthetists (and bats) inhabit
a very aural world. They are alert to signals,
perhaps inaudible to others,
which allow them to maintain complete control.*

P R E S C R I B I N G N O T E S

▼Special reporting to the CSM required.
Consult data sheet before prescribing.

Use: Induction and maintenance of general anaesthesia for surgical procedures lasting up to one hour.

Presentation: Ready to use, isotonic, aqueous emulsion containing 10mg/ml propofol in a vehicle containing soybean oil and purified egg phosphatide.

Dosage and administration: **Induction:** Titrate against response using approximately 4ml every 10 seconds in healthy adults and 2ml every 10 seconds in patients of ASA grades 3 and 4. Patients under 55 years are likely to require 2.0 to 2.5mg/kg, older patients may require approximately 20% less.

Maintenance: Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates for 10 to 20 minutes after induction. The infusion can be used undiluted or diluted with 5% Dextrose (intravenous infusion BP). Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be



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Paediatric Use: No experience in children or in mothers who are breast feeding.

Contraindications: Allergy to 'Diprivan'.

Precautions: Ampoules should be inspected before use for particulate matter and discolouration and aseptic technique followed. Discard any unused 'Diprivan' after single patient use. Do not mix prior to administration with other agents or infusion fluids other than Dextrose 5%, such dilutions should be prepared immediately before administration. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and a lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment; epilepsy; in hypovolaemic or debilitated patients; and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. Bradycardia may occur due to the lack of vagolytic activity of 'Diprivan' and administration of an anticholinergic should be considered particularly if vagal

Diprivan▼
propofol
i.v. anaesthesia

tone is dominant. Do not use in pregnancy except for termination. Discharge after general anaesthesia - allow adequate time for full recovery.

Side effects: Epileptiform movements, including convulsions and opisthotonus, have occurred rarely in a temporal relationship to 'Diprivan'. Nausea, vomiting and headache in a small proportion of patients. Very rarely, clinical features of anaphylaxis, which may include bronchospasm and erythema accompanied by hypotension, have been reported with 'Diprivan'. Pain on injection in a proportion of patients. Discolouration of urine, venous sequelae and fever are rare. Minimal evidence of excitation on induction.

Product licence number and basic NHS cost: 'Diprivan' (29/0190), £3.98 per 20ml ampoule, £9.95 per 50ml infusion vial.

'Diprivan' is a trademark.

Further information is available from
ICI Pharmaceuticals (UK), Southbank, Alderley Park, Macclesfield, Cheshire SK10 4TF.

Anaesthesia

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ANAESTHESIA: ISSN 0003-2409, Volume 45 1990, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. All advertising enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293).

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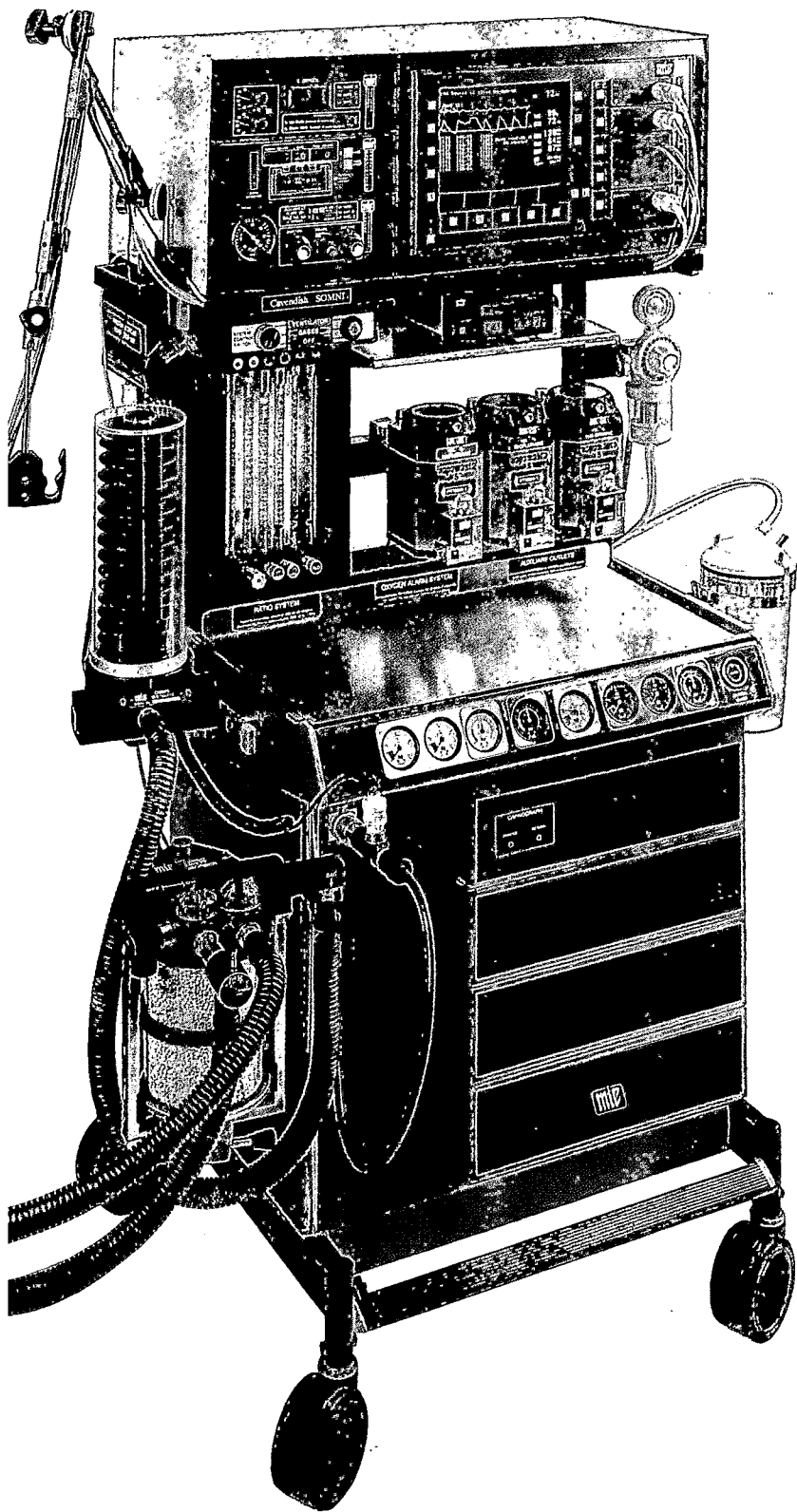
Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

Anaesthesia is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

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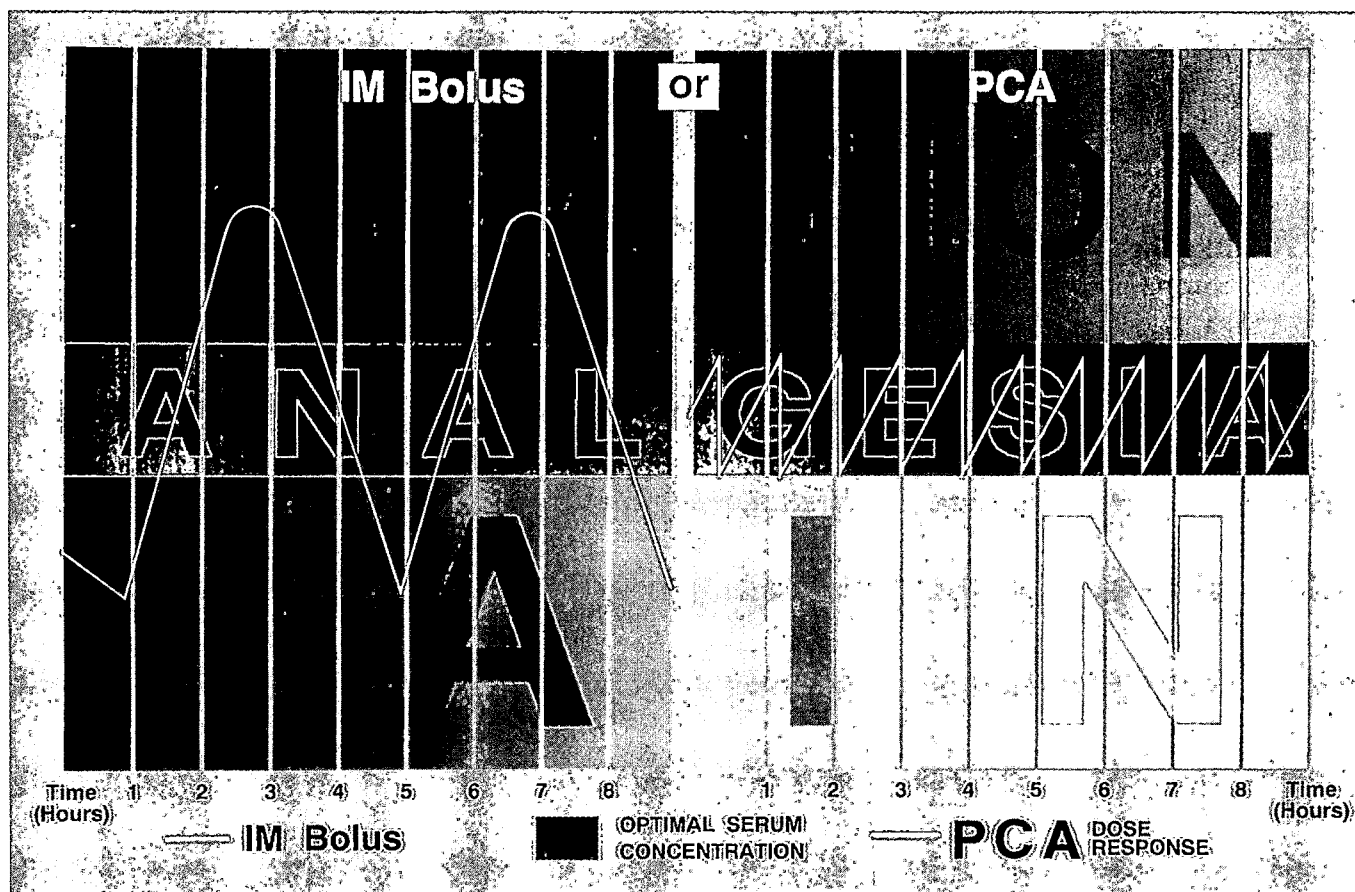
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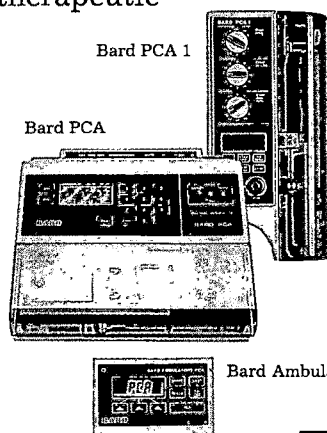
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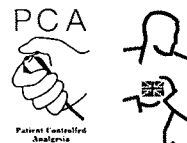


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Editorial

Safety in numbers

One of the tasks which falls to editors is the choice of subjects for leading articles. When that choice is for oneself, and the last one as Editor at that, the decision is somewhat taxing. A common trap into which one does not wish to fall is the attempt at a retrospective soliloquy. The bait is, of course, very attractive not least because all the information is available! A gaze into the future is another possibility, but we live in such changing times that there does not seem to be much point in that idea, and in any case, crystal balls are not my favourite objects.

No, a much more attractive choice is to use this opportunity as a platform to communicate something to my readers about another of my occupations. Epidemiology is the nearest word to describe this; but that is not the precisely correct term. The collection of data from a wide spectrum of practice of anaesthesia, in order to derive lessons for everyone, can help to improve the care of patients. Pooled data from many sources may be deceptive but they are superior, in at least numerical value, to the single statement of opinion of experience by one individual. We first used death in hospital as hard information about outcome (it was collected by our own clerical staff in Cardiff) and then changed to death within 6 days of an operation as the criterion for inclusion in the study in five regions of the United Kingdom. The period was extended to 30 days when surgeons joined in the *Confidential Enquiry into Perioperative Death (CEPOD)*.

Not everyone appreciates that *death* is no longer the outcome variable which is studied. Death is merely a sentinel event by which a sample of cases is obtained for study of the process of care. Another method whereby a sample of cases could be collected may be developed, but at the moment the system we have works tolerably well.

Retrospective studies, with death as the end point, suffer from many disadvantages which are well-known but these studies are not usually prospective. However, an ambitious and enterprising prospective study was prosecuted in a number of North American hospitals, and reported in part recently,¹ 4 years after the completion of the study! These results were eagerly awaited by many interested people and this delay is difficult to understand. The study was a prospective and randomised one of outcomes of four different regimens of anaesthesia for elective surgery; halothane, enflurane, isoflurane and fentanyl. There were no surprises: hypotension and bronchospasm were common with fentanyl; tachycardia with isoflurane; serious arrhythmias and slow recovery with halothane.

Early (7-day) death was rare, but hardly unexpectedly so since 91% of the 17000 patients were ASA 1 or 2, and the average age was 43 years. Nevertheless, to the writer one death in every 905 anaesthetics seems surprisingly high, not low as the investigators claim. Seven of

these 19 patients were ASA 4, nine were ASA 3 and three were ASA 2; seven (different) deaths were classified as possibly related to anaesthesia.

But, it must be understood, this lethality rate happened as a result of the method of selection of patients for study which was not random from the entire population of surgical patients.* Each subject had to be deemed suitable by the investigators for any of the agents which were studied. (Practitioners who use halothane regularly should also recall that our American colleagues claim that they seldom use this drug so it is hardly surprising that, when forced by a protocol to do so, complications occurred frequently.) Nitrous oxide and muscle relaxants were given as required.

Not only did this large study in University Hospitals fail to include all patients for surgery but it also failed to take into account, in the published analyses, the complexity of the operation or its duration. This single-minded approach may be good science and praise-worthy, and I believe it is, but it ignores the other facts behind death or morbidity associated with operations: the patient's surgical condition, the surgeon and the surgery. Anaesthetists, even anesthesiologists, cannot carp legitimately when surgeons exclude anaesthetic matters from their studies if they themselves do the same! Anaesthesia is the least important numerically and it is not the sole factor. Issues of health care are indivisible and thus *all* the relevant factors about patient management must be considered if patients themselves are to benefit.

Readers in Britain will know that the *National Confidential Enquiry into Perioperative Deaths (NCEPOD)* does include these topics in its protocol. We also need (perhaps unlike our American friends) to remember the provision of facilities and the organisation of services by departments of anaesthesia. There are two matters which are important if the unique NCEPOD is to achieve its aims. Firstly, the whole-hearted cooperation of clinicians, which currently we enjoy, needs to continue (please). Secondly, somehow our Department of Health must be encouraged to provide clinicians promptly with accurate numbers of operations and deaths so that rates of occurrence can be calculated and comparisons enabled. The latter ability is denied by the inordinate delay before the relevant data are made available.

Quantitation is difficult, but this latest project with all its (recognised) defects may yet, not only provide some

*It might be of interest to quote a few figures for comparison. The 7-day crude hospital death rate (1989) in the three University Hospitals in Cardiff after all types of surgery was 1 in 208. The age range extended from birth until the ninth decade!

answers to these fascinating problems, but also improve the quality of care in anaesthesia.

This is the last issue for which the writer is responsible as Editor. The last 8 years have passed rapidly; the experience was educative for me and, I hope, for our readers. Our authors have served us well. Long may they continue to do so. The time has now come for me to hand over to my successor, Dr Maldwyn Morgan, and to wish him and *Anaesthesia*, well.

J. N. LUNN

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Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; **1**: 432 5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Gastro-oesophageal reflux during elective laparoscopy

C. J. ROBERTS AND N. W. GOODMAN

Summary

An oesophageal pH electrode was used to record gastro-oesophageal reflux in 73 women who had elective laparoscopy for various gynaecological procedures. No refluxes were recorded during the 63 procedures from which results could be analysed; the upper 95% confidence limit from this observation is 3 in 63 (4.8%). Two of the excluded women refluxed during episodes of hiccough that occurred shortly after induction of anaesthesia. Tracheal intubation may be required during laparoscopy, although the need to protect against the possibility of aspiration of gastric contents may not be a valid reason unless, with the same logic, it is suggested that all patients who hiccough should be intubated.

Key words

Complications; aspiration.

Surgery; laparoscopy.

It is commonly thought that laparoscopy increases the likelihood of regurgitation of gastric contents. Reasons given include the lithotomy position, head-down tilt, the surgeon pressing on the abdomen and insufflation of the peritoneal cavity. Duffy¹ measured the pharyngeal pH at the end of the procedure; in two of 93 patients who had undergone elective laparoscopy the pharyngeal contents were acid. Carlsson and Islander² used a similar technique and showed that regurgitation occurred in 20% of emergency laparoscopies.

A continuous intra-operative recording of oesophageal pH in the present study was made with an antimony pH electrode placed in the distal oesophagus during elective laparoscopies.

Methods

The study was approved by the hospital ethics committee. Seventy-five consecutive women who required anaesthesia for elective laparoscopy were approached for the study and all but two gave informed consent.

The patients were ASA 1 or 2, aged 18 to 62 years, and weighed between 45 and 86 kg. The ratio of weight to the square of height was used as a measure of obesity: the mean (range) was 23.7 (18.3 to 33.7) kg/sq m (mild obesity is 30 kg/sq m). Four patients gave a history of occasional

reflux after meals. All patients were fasted by mouth for at least 8 hours, except that temazepam 20 mg was offered as premedication and accepted by 12 patients.

Anaesthesia was induced with propofol (2.5 mg/kg) and fentanyl (2.3 µg/kg) and vecuronium (0.08 mg/kg) was given to provide muscular relaxation. The patients' lungs were ventilated by hand with 67% nitrous oxide in oxygen, with 1% isoflurane or enflurane, before laryngoscopy and intubation of the trachea. Anaesthesia was maintained with inhalational agents delivered by mechanical ventilation.

Oesophageal pH was measured with a monocrystalline antimony pH electrode (Synectics Medical Ltd) calibrated before each set of measurements with standardised solutions of buffer at pH 1 and 7. The electrode is enclosed in the tip of a 2-mm soft plastic tube, which was inserted under direct vision at laryngoscopy and advanced until gastric pH was recorded. It was then withdrawn to the gastro-oesophageal junction, recognised by an abrupt increase in pH, and finally positioned 4 to 5 cm above the junction. Any subsequent decrease in the measured pH to 4 or less indicated reflux.

The data were stored in a solid state memory and subsequently transferred to a computer to be analysed with Gastrosoft software (Digitrapper and Oesophogram: Synectics Medical Ltd). This equipment is the same as was used in this department in a previous study.³

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Accepted 4 April 1990.

Results

Sixty-three of the 73 patients who gave consent, including the four with a history of occasional reflux, gave satisfactory results; the other 10 had to be excluded. The electrode would not pass into the stomach in three patients; two patients had gastric contents that were less acid than pH 4.5; two other patients hiccupped after induction and their oesophageal contents became acid at that stage, which precluded any further measurement of reflux; in one patient, the surgeons decided to do a laparotomy instead of a laparoscopy; and for two patients, failure of the battery in the solid state memory meant that their data were lost.

Details of the operations are shown in Table 1. Head-down tilt was 15–20°; abdominal pressure during insufflation was 1.06–1.3 kPa. Gastric pH was 1.97 (SD 0.38) and oesophageal pH 5.91 (SD 0.54) in the 63 patients studied successfully. There was no evidence of gastro-oesophageal reflux during laparoscopy. With an observed incidence of no refluxes on 63 occasions, the upper 95% confidence limit of the true incidence of occurrence is 3 in 63, or 4.8%.⁴

Discussion

In a prospective series of 50 048 laparoscopies studied by a working party,⁵ only one patient regurgitated and this occurred at induction; this rate of 0.002% is much less than the 2% reported by Duffy.¹ The overall incidence of regurgitation during various forms of general anaesthesia was reported as 0.05% in 185 358 procedures by Olsson and co-workers⁶ and 9% in 734 procedures by Blitt and co-workers.⁷

Writers of recent letters to *Anaesthesia* have argued both for and against the need for tracheal intubation in patients who have laparoscopy under general anaesthesia.^{8–10} Neuromuscular relaxation and tracheal intubation have risks and are associated with an increased risk of the circumstance against which they protect, namely regurgitation.⁷ They also cause greater morbidity in the early postoperative period than does anaesthesia with a mask.¹¹ If we choose to protect patients against one set of risks, we must be reasonably sure we are not imposing a worse set.

It sounds logical to suppose that laparoscopy predisposes to regurgitation but, as with many decisions and treatments in clinical medicine, we must be wary of acting as though this assumption were true. Hamilton wrote,¹² 'It is thus legitimate to make the best assumption possible and base our predictions on these assumptions. Absolutely nothing is wrong with this course of action... However... we must not believe or pretend or teach or behave as if our assumption is fact.'

We saw no episodes of regurgitation during 63 elective laparoscopies in unselected patients, although none of them was grossly obese or admitted to more than occasional symptoms of reflux after meals. The lungs were ventilated by mask before laryngoscopy in all the patients.

The physiology of the gastro-oesophageal junction is complex. The site of insertion of the upper leaf of the gastrophrenic ligament¹³ and the length of the lower oesophageal sphincter within the abdominal cavity¹⁴ may be important in the maintenance of competence of the gastro-oesophageal sphincter. Jones and co-workers¹⁵ reported an adaptive increase in the pressure of the lower oesophageal sphincter in response to increases in abdominal pressure caused by insufflation. Insufflation may flatten the intra-abdominal oesophagus, adding to the overall barrier pressure of the sphincter. In addition, the passive diaphragmatic movements of controlled ventilation produce smaller oscillations of lower oesophageal sphincter pressure than occur during the intrinsic diaphragmatic contractions of spontaneous breathing.¹⁶

However, although it is possible to suggest ways in which insufflation will not increase the likelihood of regurgitation, what matters clinically is not the demonstration of normal or abnormal physiological mechanisms but whether precautions, which have their own risks, should be taken.

We cannot make a firm conclusion from studying 63 patients, all we can do is put forward our observations for consideration. Our upper 95% confidence limit on the true incidence in similar patients is one in 21, just under 5%, but the lower confidence limit is zero; there is an observed incidence of 2 in 253 if we add our figures to those of others.^{1,2} Not everyone in whom there is reflux of acid into the lower oesophagus will actually regurgitate, or subsequently aspirate, so what is an acceptably low incidence of reflux? In other words, what incidence would be acceptable to anaesthetists for not requiring the protection of the trachea by intubation? On the similar subject of the need for precautions in obstetric anaesthesia, Hamilton¹² pointed out that since Mendelson's original observations there has been much work on pH and gastric contents and much pontification but, 'all surveys known to me since institution of this prophylactic prescription state (that) the incidence of aspiration as a major complication of obstetric anaesthesia has not decreased.'

Two of the patients hiccupped during induction of anaesthesia, and both refluxed with the hiccupps. The incidence of regurgitation was recorded during vaginal termination of pregnancy in another study in this unit (unpublished observations); again, the only episode of reflux in 41 patients was recorded after hiccup. On this evidence, with three out of three patients who had reflux

Table 1. Operative procedure, duration; mean and (range) of lithotomy and head-down positions, and duration; mean and (range) of insufflation, during 63 elective laparoscopies.

Procedure	Number	Time (minutes)		
		Lithotomy	Head-down	Insufflation
Sterilisation	24	14 (9–21)	7 (2–17)	8 (4–17)
Hydrotubation	16	15 (11–24)	8 (3–22)	9 (4–19)
Diagnostic	13	15 (9–27)	14 (5–59)	9 (4–20)
Oocyte recovery	10	—	38 (19–60)	39 (21–63)

during hiccough, it would be more logical to suggest any patient who hiccoughs must have her trachea intubated than to suggest this course of action just because a patient is undergoing laparoscopy. We doubt there are many anaesthetists who would do this, and we are not advising it, merely pointing out the logic of the situation.

The working party of the confidential enquiry into gynaecological laparoscopy recommended that general anaesthesia for the procedure should be by tracheal intubation, controlled ventilation, and good relaxation.⁵ We are not suggesting these patients do not need tracheal intubation; we are suggesting it may not be true that they are particularly at risk of aspiration. Intubation may still be necessary for other reasons, associated with adequacy of ventilation, hypercapnia, or to give the surgeon better operating conditions.

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Arterial oxygen saturation during induction of anaesthesia

C. M. THORPE AND I. S. GAUNTLETT

Summary

Three groups of 10 ASA 1 patients were studied to determine the incidence of hypoxaemia (oxygen saturation $\leq 90\%$) using pulse oximetry during induction of 'mask' anaesthesia, and whether simple oxygenation techniques could prevent its occurrence. We also surveyed all anaesthetists in three major hospitals to ascertain their techniques for this method of anaesthesia. Anaesthesia was induced in all patients with thiopentone and maintained with nitrous oxide and isoflurane. The first group received 33% oxygen in nitrous oxide as carrier gases, a second group a few normal breaths of 100% oxygen during thiopentone administration followed by 33% oxygen in nitrous oxide, while a third group received 100% oxygen after loss of eyelash reflex until spontaneous breathing was established. No patient received positive pressure ventilation before spontaneous breathing was established. Six of the 10 patients in the first group became hypoxaemic compared to none in the second group, and three patients became hypoxaemic in the third group. Thirty-seven percent of anaesthetists who responded to the survey either did not apply positive pressure ventilation before establishment of spontaneous breathing, or only did so if apnoea was prolonged. Only one anaesthetist fully pre-oxygenated patients lungs. We conclude that to avoid the likely occurrence of hypoxaemia during induction of mask anaesthesia, a minimum of a few breaths pre-oxygenation is necessary.

Key words

Induction; anaesthesia.

Equipment; pulse oximeter.

Mask anaesthesia, whereby anaesthesia is administered via a mask and semiclosed breathing system with the patient breathing spontaneously, is widely used in the UK. Administration of general anaesthesia by mask during spontaneous ventilation accounted for 28.8% of cases in a recent survey of anaesthetic practice in a wide selection of hospitals.¹ It is our practice to induce this method of anaesthesia with an intravenous induction agent, and administer a mixture of 66% nitrous oxide (N_2O) in oxygen (O_2) with a volatile agent. We wait for breathing to resume without applying positive pressure ventilation if transient apnoea occurs; no pre-oxygenation is used. We use pulse oximetry both during the induction and maintenance of anaesthesia as recommended by the Association of Anaesthetists.² We observed that in a considerable number of patients measured O_2 saturation (SpO_2) decreased to $\leq 90\%$, which, if accompanied by other complications of induction such as laryngospasm, might proceed to severe hypoxia.

The failure to pre-oxygenate patients' lungs for routine general anaesthesia is common in Great Britain; in the Survey of Anaesthetic Practice¹ only 21% of anaesthetics

were preceded by pre-oxygenation; 9% were for emergencies. Thus it is possible that, if our approach to induction of anaesthesia (with no attempt to control ventilation before breathing resumes) is common, substantial numbers of patients may be exposed to hypoxaemia during routine induction of anaesthesia.

We surveyed all anaesthetists in three major hospitals in Bristol to estimate how common our technique is for induction. We then undertook a study to investigate the incidence of hypoxaemia during induction using this technique, and to assess whether each of two manoeuvres (administration of 100% O_2 for a few breaths either before induction or after loss of eyelash reflex) were effective in reducing that incidence.

Methods

Oxygenation study

We studied 30 ASA 1 patients undergoing short day-case procedures, after approval of our local ethics committee, and with informed consent. Those patients with a history

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Accepted 30 January 1990.

of smoking, recent respiratory infections or obesity were excluded from the study. Patients were allocated randomly to one of three groups and were unpremedicated. A Lack breathing system and facemask administering fresh gas at 6 litres/minute was used for all patients. Induction of anaesthesia was with intravenous thiopentone given over 10–20 seconds. Group A ($n = 10$) with no pre-oxygenation received 66% N₂O in oxygen with 2% isoflurane after loss of eyelash reflex. Group B ($n = 10$) received 100% O₂ (6 litres/minute) via the facemask just as injection of thiopentone was started and were told to breathe normally. These patients received approximately 4–5 breaths 100% O₂ before loss of eyelash reflex, then 66% N₂O in oxygen with 2% isoflurane after loss of eyelash reflex. Group C ($n = 10$) were not pre-oxygenated but received 100% oxygen (6 litres/minute) via the facemask immediately after loss of eyelash reflex and this was continued until normal breathing was established, after which they received the same anaesthetic mixture as groups A and B. All anaesthetics were administered by one of the authors (I.S.G.).

A Nellcor pulse oximeter (model N100) set in Mode 2

(therefore updating every 2 seconds) was used with a standard finger probe to monitor SpO₂ continuously. Alarms were suspended and the audible tone of the oximeter switched off. Recordings were made at 10-second intervals and, in addition, the lowest saturation achieved was noted. The anaesthetist was blinded to the SpO₂ value during induction, but would be informed if it decreased to below 85% and the study would be terminated. This situation did not occur. Hypoxaemia was prospectively defined as an oxygen saturation of 90% or less.^{3–6} The difference between groups A and B and A and C was tested using one-tailed Fisher's exact probability test. The difference between the groups for patient characteristics was tested using the Mann-Whitney *U*-test. A *p* value < 0.05 was considered significant.

Survey of anaesthetic technique

Ninety-three questionnaires (Fig. 1) were sent to all anaesthetists of all grades in three large hospitals in Bristol

Hospital:	BRI	Frenchay	Southmead		
Status:	Consultant	Senior Registrar	Registrar	SHO	Other

A young ASA 1 patient is to undergo a short surgical procedure as a day patient for which you plan to give an anaesthetic roughly as follows. Anaesthesia is induced with an intravenous anaesthetic agent. Following this the patient breathes spontaneously a mixture of 66% nitrous oxide, 33% oxygen and a volatile anaesthetic agent. No muscle relaxants are given and tracheal intubation is not planned. Please mark which of the following would most accurately reflect your *usual* practice. If you never give this type of anaesthetic, please indicate so on this form and return it anyway.

Induction agent: thiopentone propofol methohexitone other
(give approximate dose for a 70-kg man).

Analgesia:
If the procedure is painful (e.g. excision lipoma on chest), which analgesic would you give *prior* to induction:

none fentanyl alfentanil pethidine papaveretum other
(give approximate dose for a 70-kg man).

If the procedure is not painful (e.g. D & C), which analgesic would you give *prior* to induction:

none fentanyl alfentanil pethidine papaveretum other
(give approximate dose for a 70-kg man).

Pre-oxygenation: none few deep breaths full 3-5 minutes other

Circuit: Magill Lack Bain Other

Following induction with an intravenous agent, do you:

1. Apply mask with a mixture of 66% nitrous oxide, 33% oxygen and a volatile anaesthetic and wait for breathing to recommence (if apnoea occurs)?
2. Apply mask with 100% oxygen only and wait for breathing to recommence (if apnoea occurs)?
3. Apply mask with 100% oxygen and a volatile anaesthetic and wait for breathing to recommence (if apnoea occurs)?
4. Apply mask with a mixture of 66% nitrous oxide, 33% oxygen and a volatile anaesthetic and control ventilation until breathing recommences (if apnoea occurs)?
5. Apply mask with 100% oxygen only and control ventilation until breathing recommences (if apnoea occurs)?
6. Apply mask with 100% oxygen and a volatile anaesthetic and control ventilation until breathing recommences (if apnoea occurs)?
7. Other (please specify).

Fig. 1. Questionnaire on anaesthetic practice sent to all anaesthetists in Bristol hospitals.

Table 1. Patient data.

Group A. No added oxygen (66% N ₂ O in oxygen)								
Patient	Age (years)	Sex	Weight (kg)	Thiopentone (mg/kg)	Apnoea (seconds)	Pre-induction % SpO ₂	Lowest % SpO ₂	Hypoxaemia* (yes/no)
1	22	female	62	4.8	30	100	89	yes
2	20	female	47	7.4	—	98	90	yes
3	32	female	62	5.6	40	98	89	yes
4	19	male	85	5.9	15	98	95	no
5	39	female	60	6.7	40	100	96	no
6	23	female	46	6.5	20	98	97	no
7	20	female	55	6.4	25	99	90	yes
8	18	female	64	6.3	20	100	86	yes
9	44	male	70	7.1	20	99	94	no
10	26	female	58	7.8	80	100	90	yes
Group B. Few normal breaths pre-oxygenation before induction								
1	20	male	64	7.0	30	99	99	no
2	29	female	71	5.6	50	98	98	no
3	26	female	57	6.1	—	97	97	no
4	29	female	50	8.0	90	100	100	no
5	23	female	66	5.7	40	99	98	no
6	27	female	—	—	30	98	98	no
7	27	female	58	5.2	40	100	100	no
8	25	female	53	6.1	30	99	98	no
9	28	male	80	6.2	30	100	100	no
10	34	female	75	5.0	30	100	100	no
Group C. Oxygenation after loss of eyelash reflex until breathing re-established								
1	41	female	55	6.4	10	97	89	yes
2	18	female	56	4.9	20	100	93	no
3	18	female	55	5.9	40	100	88	yes
4	21	female	62	5.6	20	99	99	no
5	23	male	70	—	45	97	97	no
6	21	male	—	—	40	98	87	yes
7	21	female	72	6.9	20	100	98	no
8	32	male	85	5.9	45	97	97	no
9	28	male	75	8.0	30	97	92	no
10	20	male	68	7.4	90	100	92	no

* $p < 0.05$ group A compared with group B (Fisher's exact probability test, one-tailed).

(Bristol Royal Infirmary, Southmead and Frenchay Hospitals); 71 replies were received, a response rate of 76%. Those surveyed were asked to choose, from a selection of techniques, the induction technique that most closely resembled their own.

Results

Data for all patients are given in Table 1. There were no differences between the groups in age, weight, sex, dose of thiopentone/kg or duration of apnoea. All patients had an initial oxygen saturation of 96% or greater. Six patients in group A and three in group C were hypoxaemic, whereas no patients were hypoxaemic in group B. The difference between groups A and B was significant, whereas there was no difference between groups A and C.

Results of survey

The results of the survey are presented in Table 2. Only data relevant to this study are presented. Seventeen percent of those who responded stated that they routinely used a similar technique to ours, induction of anaesthesia intravenously, application of the mask and waiting for breathing to resume without intervention. A further 20% stated that they used the above technique, but volunteered that if

apnoea lasted 'too long' they would apply positive pressure ventilation with 66% N₂O in oxygen until breathing resumed. No one specified what duration of apnoea constituted 'too long'. Thus 37% undertook induction of anaesthesia by the same technique as that administered to group A. Only one respondent practised full pre-oxygenation (approximately 2 minutes) while a further four gave a 'few breaths'.

Table 2. Survey of anaesthetic induction technique. Number of questionnaires sent = 93; number of replies = 71; response rate = 76%.

Induction technique	Number	Percent
66% N ₂ O in O ₂ with volatile agent; no IPPV	12	17
66% N ₂ O in O ₂ with volatile agent; + IPPV	42	59
66% N ₂ O in O ₂ with volatile agent; IPPV if long apnoea	14	20
Other	3	4
Pre-oxygenation	Number	Percent
None	62	87
Few breaths	4	6
2 minutes	1	1
No comment	4	6

Discussion

Our knowledge of what happens to patients' oxygenation peri-operatively has greatly increased since the advent of the pulse oximeter into clinical use in the past few years. The occurrence of hypoxaemia in the postoperative period in patients to whom added oxygen is not given is well known,³⁻⁹ as is the benefit of oxygen therapy.^{6,8} Further studies have described the incidence of hypoxaemia intra-operatively.^{10,11} In addition to the study by Cote *et al.*⁹ who examined the whole peri-operative period, there were two recent studies that documented the occurrence of hypoxaemia during induction in children,^{12,13} but we found no recent data that related to hypoxaemia during normal induction of anaesthesia in adults. Dillon and Darsie in 1955¹⁴ demonstrated the occurrence of arterial O₂ desaturation after induction of anaesthesia in patients who had not been pre-oxygenated. However, the patients were given intravenous pethidine 100 mg before thiopentone injection, which would be expected to increase any ventilatory depression from thiopentone and thus make hypoxaemia worse. Weitzner *et al.* in 1959¹⁵ demonstrated profound arterial O₂ desaturation in patients rendered apnoeic after hyperventilation with air (values as low as 49% were recorded), and this has more recently been confirmed by Drummond and Park.¹⁶ However, in both of these studies patients were given suxamethonium to produce apnoea, and no anaesthetic gases were administered, so there was no possibility of them taking any breaths during this time. Their approach, whilst providing valuable information, does not bear any relation to any commonly used induction techniques. The duration of detectable apnoea was between 10 and 60 seconds in our patients who became hypoxaemic, yet the lowest recorded SpO₂ was 86%.

Two factors have probably led to the paucity of studies into oxygen saturation during anaesthetic induction. Firstly, most studies using pulse oximetry come from North America where this monitor has been used more widely and for a longer period. In North America most patients are routinely pre-oxygenated^{17,18} before induction, and the use of 'mask anaesthesia' is probably less common than in the United Kingdom. Secondly, although pulse oximetry has been available in the United Kingdom for several years, monitoring of oxygenation during induction is rare and pre-oxygenation not routine practice.¹ The lack of routine use of pre-oxygenation is confirmed by the results of our survey. The use of oxygenation monitoring during induction may increase since publication of guidelines by the Association of Anaesthetists of Great Britain and Ireland.² The high incidence of hypoxaemia from this technique suggests that a considerable number of patients are put at risk during short, routine anaesthetics if Bristol anaesthetists are representative of national practice. An arterial oxygen saturation of 86% does not represent a life threatening event, but should some other complication of induction (e.g. laryngeal spasm) supervene, the results could be catastrophic. We have demonstrated that the technique of induction used for group B, which involved the patient taking a few normal breaths of O₂, significantly reduces the incidence of hypoxaemia. We deliberately avoided asking patients to take deep breaths (or vital capacity breaths) that have previously been shown to be effective,^{18,19} since we wished to avoid hyperventilation, which might prolong the duration of apnoea.

We have demonstrated a high incidence of hypoxaemia in ASA I patients undergoing short 'mask' anaesthesia with no pre-oxygenation and have shown that some patients become hypoxaemic as soon as 10 seconds after onset of apnoea, a period that must be easily achieved even if positive pressure ventilation is applied shortly after induction. A short period of pre-oxygenation can reduce the incidence of hypoxaemia. Our results suggest that all patients undergoing 'mask' anaesthesia may benefit from pre-oxygenation, and consideration should be given to administering 100% O₂ for a few breaths before induction of anaesthesia.

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The effects of midazolam on cerebral blood flow and oxygen consumption

Interaction with nitrous oxide in patients undergoing craniotomy for supratentorial cerebral tumours

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Summary

Cerebral blood flow and the cerebral metabolic rate of oxygen were measured in 30 patients during craniotomy for supratentorial cerebral tumours by a modification of the Kety–Schmidt technique using Xenon 133 intravenously. Anaesthesia was induced with midazolam 0.3 mg/kg, fentanyl and pancuronium, and maintained with midazolam as a continuous infusion, fentanyl, pancuronium and nitrous oxide in oxygen or oxygen in air. The concentration of midazolam in the blood of 10 patients was about 300 ng/litre during two measurements; the patients' lungs were ventilated with N₂O in oxygen. The concentration of midazolam in the blood of another 10 patients was doubled to about 600 ng/litre during the second flow measurement; the patients' lungs were ventilated with N₂O/O₂. The concentration of midazolam in the blood of the third group of 10 patients was doubled to 600 ng/litre during the second flow measurement; the patients' lungs were ventilated with oxygen in air. No relationship was found between the dose of midazolam and cerebral blood flow or oxygen consumption. Nitrous oxide in combination with midazolam also had no effect on these variables.

Key words

Hypnotics; benzodiazepines, midazolam.
Brain; blood flow, oxygen consumption.

Studies of the effects of midazolam anaesthesia on cerebral blood flow (CBF) and the cerebral metabolic rate of oxygen (CMRO₂) are few and conflicting. It is suggested in animal work that midazolam produces a dose-related decrease of cerebral metabolism, blood flow and encephalographic activity.^{1–3} A human study concludes that midazolam does not produce a significant decrease in CBF;⁴ however, no control group was included. A study concludes in human volunteers that doses of 0.15 mg/kg produce a significant decrease in CBF, but PaCO₂ and mean arterial blood pressure (MAP) were not constant.⁵

Nitrous oxide in rats has little effect on CBF and CMRO₂.⁶ In the same animal, another study concludes that cerebrovascular response to midazolam infusion is not altered by nitrous oxide, but CMRO₂ decreased significantly more during nitrogen/oxygen inhalation compared with nitrous oxide/oxygen inhalation, which suggests that nitrous oxide may stimulate brain metabolism during midazolam infusion.³

The purpose of the present investigation was to measure CBF and CMRO₂ at two different concentrations of midazolam, and to see if there was a dose-related relationship.

Furthermore, the effect of N₂O on CBF and CMRO₂ during midazolam infusion was also investigated.

Methods

Patients

CBF and CMRO₂ were measured twice in 30 patients with supratentorial cerebral tumours. The median age of the patients was 48 (range 21–74) years. Twenty-two males and eight females were included. There were no significant differences between the groups with regard to sex, age, weight, histological diagnosis and tumour size. All the patients gave informed consent and the study was approved by the local ethics committee, and was in accordance with the Helsinki 11 declaration.

Only patients with a midline shift of < 10 mm estimated by CT scanning were included in the study. Patients undergoing treatment for heart disease, hypertension or chronic pulmonary diseases were not studied. All the patients were

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Accepted 21 March 1990.

awake before operation, were orientated and without major neurological failure. They were being treated with dexamethasone 6 mg \times 4 daily.

Anaesthesia

The patients were premedicated with oral diazepam 10–20 mg 2 hours before induction. Anaesthesia was induced, after pre-oxygenation, with midazolam 0.3 mg/kg, fentanyl 4 μ g/kg and pancuronium 0.15 mg/kg. The patients were manually ventilated until total paralysis. Approximately one minute before tracheal intubation intravenous lignocaine 1.5 mg/kg was given. Anaesthesia was maintained, after tracheal intubation, with midazolam and 66% nitrous oxide in oxygen supplemented with fentanyl 4 μ g/kg/hour and pancuronium 1–2 mg sufficient to provide relaxation estimated by train-of-four stimulation. The patients' lungs were ventilated during anaesthesia with a Servo ventilator to an end-expiratory CO₂ of 4 kPa, and by analyses of arterial blood gases (ABL-3, Radiometer, Copenhagen, Denmark). Rectal temperature was measured repeatedly and mean arterial blood pressure (MAP) was continuously directly monitored by an intra-arterial line.

The scalp was infiltrated with bupivacaine 0.25% before skin incision.

The patients were randomly divided into three groups. The midazolam infusion rate remained constant in group 1 ($n = 10$) during the two CBF measurements. After induction with midazolam 0.3 mg/kg, the infusion rate was 0.68 (mg/kg)/hour for 15 minutes followed by 0.125 (mg/kg)/hour. This infusion regimen was expected to result in a steady-state concentration of about 300 ng/ml from 45 minutes onwards.⁷ The patients' lungs were ventilated with 66% nitrous oxide. In group 2 the midazolam infusion rate was doubled after the first CBF measurement. The patients were given a bolus of 0.3 mg/kg midazolam after the first CBF measurement and the infusion rate was increased to 0.25 (mg/kg)/hour. This infusion regimen was expected to result in a steady-state concentration of about 600 ng/ml. The patients' lungs were ventilated with nitrous oxide in oxygen. The midazolam infusion rate in group 3 was the same as in group 2. The patients' lungs were ventilated with nitrous oxide in oxygen during the first CBF measurement, but during the second measurement the patients were ventilated in the absence of nitrous oxide.

Mannitol

Mannitol 0.5 g/kg was administered intravenously after the first CBF flow measurement, and 10–15 minutes before the surgeon started to open the dura mater. The mannitol infusion was carried out in 5 minutes and finished before the second flow measurement.

Measurement of CBF and CMRO₂

A catheter was introduced percutaneously into the internal jugular vein, after induction of anaesthesia; the tip of the catheter was placed at the base of the skull. Xenon¹³³ (3 mCi dissolved in 30 ml saline) was injected intravenously during a period of 20 minutes. Blood samples of 2 ml were withdrawn from the arterial and internal jugular vein catheters at exact time intervals during a 30-minute desaturation period. The sample radioactivity was counted in a

well counter (Berthold LB MAG 510). CBF was calculated according to the height-over-area formula of Kety and Schmidt:⁸

$$CBF = H/Area \times \Lambda \text{ ml/100 g/min}$$
$$CMRO_2 = AVDO_2 \times CBF$$
where AVDO₂ is the arteriovenous difference of oxygen. This intravenous modification of the classical Kety–Schmidt method⁸ was described recently.⁹ The AVDO₂ was calculated as the difference in oxygen content between arterial and venous blood. Oxygen saturation and oxygen tension were measured by ABL-3 (Radiometer, Copenhagen, Denmark). The haemoglobin was measured separately. The AVDO₂ was measured in duplicate at each CBF measurement.

The first CBF measurement was performed at a median of 80 minutes (range 52–135 minutes) after induction and before incision. The second CBF measurement was performed exactly one hour later, median 140 minutes (range 112–195 minutes) after induction and after the dura was opened.

Assay of midazolam in plasma

Analyses were measured by gas-chromatography and performed by Roche Basle. Five millilitres of arterial blood were withdrawn, and the plasma stored at -20°C , at the same time as each CBF measurement (5 minutes before and after desaturation of xenon¹³³).

Statistical analysis

Median and range were calculated. The Wilcoxon test was applied for paired data, Mann–Whitney for unpaired data. $p < 0.05$ was considered significant.

Results

The first CBF measurement averaged 28 (ml/100 g)/minute in group 1, and the second 23 (ml/100 g)/minute. In group 2 the first CBF measurement averaged 23 (ml/100 g)/minute, and the second 28 (ml/100 g)/minute. In group 3, the first CBF measurement averaged 24 (ml/100 g)/minute, and the second flow 26 (ml/100 g)/minute. No significant differences were found within or between the groups. The PaCO₂ and MAP were constant during the flow measurements. There was no significant difference between the three groups in the concentration of midazolam during the first CBF measurement. The concentration of midazolam during the second flow measurement increased significantly according to the trial design in groups 2 and 3, and remained unchanged in group 1 (Table 1). The results are shown in Table 2. There was no significant difference in AVDO₂ and CMRO₂ between the three groups during the two flow measurements.

Discussion

Global CBF and CMRO₂ were measured in this study peroperatively during craniotomy for supratentorial cerebral tumours. Regional CBF differences in studies of cerebral tumours were observed close to the tumour⁸ as well as remote from it.⁹ The influence of the tumour flow on global cerebral flow is unclear. However, in the present study the flow measurement was performed in patients with small cerebral tumours with no great midline shift. Consequently, we assumed that the global CBF and CMRO₂ values

Table 1. Midazolam concentration in blood (ng/ml).

	CBF1	CBF2	
Group 1; median (range)	321 (193–443)	281 (191–421)	ns
Group 2; median (range)	375 (240–500)	595 (375–830)	p < 0.05
Group 3; median (range)	280 (190–452)	665 (455–825)	p < 0.05
	ns	p < 0.05	

ns, not significant.

obtained were dominated by the large preponderance of normal brain tissue.

The cerebral blood flow was measured using an intravenous modification of the inhalation method described by Kety and Schmidt.¹⁰ The validity of this technique for CBF measurements was tested in awake patients with supratentorial cerebral tumours.¹¹ The values found correspond to values found in normal man, and argue against a major influence of a tumour on the global CBF. Furthermore, this technique has produced reliable results in repeated CBF studies.^{12–16}

Mannitol caused an increase in CBF 10 to 20 minutes after a bolus dose,^{17–19} and a variable increase in CMRO₂,^{18,19} so it was decided to prescribe mannitol treatment to all patients in the study. Therefore, the effects of mannitol on cerebral circulation and metabolism should be comparable in the three groups, and only the concentration of midazolam should influence cerebral circulation and metabolism. A dose of 0.5 g/kg mannitol was chosen to secure brain relaxation before opening of the dura. Mannitol infusion before the second flow measurement might have influenced the CBF and CMRO₂ measurements. However, expected increases in flow and metabolism during unchanged midazolam concentration were not observed.

A recent study indicates²⁰ that the increase in CBF by mannitol is effected by a decrease in blood viscosity. However, in another study the same authors have argued that if cerebral autoregulation is intact the decrease in

blood viscosity might be balanced by an increased vasoconstriction in cerebral vessels.²¹

Several studies in patients with cerebral tumours indicate global or regional loss of cerebral autoregulation.^{8,9} Moreover, studies during neurolept anaesthesia and halothane anaesthesia in patients with supratentorial tumours subjected to craniotomy indicate abolished cerebral autoregulation.²²

Work in dogs² concluded that midazolam produced a dose-related decrease in CBF and CMRO₂, but not to the same degree as other intravenous and volatile anaesthetics. Another study in the same animal¹ indicated that small doses (0.2 mg/kg) of midazolam did not decrease CMRO₂, but did decrease CBF. Higher doses (10 mg/kg) resulted in dose-related decreases in CMRO₂. In human volunteers⁵ midazolam 0.15 mg/kg produced a 33% decrease in CBF and a 40% increase in cerebral vascular resistance; however, CMRO₂ was not measured, and the PaCO₂ and MAP were not constant during the study. In another human study⁴ little or no change in CBF was observed after midazolam injection during midazolam–fentanyl–N₂O anaesthesia. In the present study the use of nitrous oxide did not influence CBF and CMRO₂. These findings are in accordance with experimental studies in rats.⁶

In rats subjected to³ intravenous infusion of midazolam, a modest cardiovascular depression and a dose-related decrease in CBF and CMRO₂ were observed.³ We found no significant relationship between concentration of midazolam, cerebral circulation and oxygen consumption. Thus,

Table 2. Time, PaCO₂, MAP, temperature, CBF, AVDO₂ and CMRO₂ in patients subjected to craniotomy for supratentorial cerebral tumours during midazolam anaesthesia; median and (range).

	Time (minutes)	PaCO ₂ (kPa)	MAP (mmHg)	Temperature °C	CBF (ml/100 g/m)	AVDO ₂ (vol %)	CMRO ₂ (ml O ₂ /100 g/m)
<i>Group 1</i>							
CBF1	83 (52–135)	4.42 (3.8–5.1)	82 (60–100)	35.9 (35–37)	28 (16–47)	8.9 (7.9–11.0)	2.7 (1.4–4.2)
CBF2	60 (60–60)	4.28 (3.7–4.8)	77 (60–103)	35.8 (35–37)	23 (16–42)	10.1 (7.1–12.0)	2.4 (1.2–4.4)
<i>Group 2</i>							
CBF1	85 (65–115)	4.05 (3.6–4.8)	78 (65–110)	35.7 (35–37)	23 (16–45)	8.9 (3.8–14.2)	2.1 (1.1–3.2)
CBF2	60 (60–60)	3.83 (3.5–4.8)	82 (75–91)	35.7 (35–36)	28 (14–42)	6.7 (3.6–12.9)	1.7 (0.9–3.8)
<i>Group 3</i>							
CBF1	75 (60–83)	4.50 (3.5–5.1)	76 (64–112)	36.2 (35–37)	24 (21–36)	9.4 (5.3–13.3)	2.3 (1.6–3.0)
CBF2	60 (60–60)	4.2 (3.2–4.7)	83 (65–108)	36.1 (35–37)	26 (16–41)	8.6 (4.6–14.8)	2.3 (1.2–3.7)
	ns	ns	ns	ns	ns	ns	ns

ns, not significant.

midazolam differs from other intravenous hypnotics including althesin²³ and etomidate,¹² where a significant dose-response relationship was found. Nevertheless, we produced excellent conditions during surgery, and an uncomplicated postoperative recovery. Furthermore, the study indicates that it is possible to avoid nitrous oxide during midazolam anaesthesia without any significant influence on CBF and CMRO₂.

Acknowledgments

The authors thank Dr L. Kirkegaard for assistance in the statistical analysis.

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Obstetric epidural analgesia with mixtures of bupivacaine, adrenaline and fentanyl

G. YAU, M. A. GREGORY, T. GIN AND T. E. OH

Summary

We performed a double-blind comparison of six solutions for epidural analgesia in 90 healthy Chinese women with uncomplicated pregnancies. Patients were randomly allocated to receive 10 ml bupivacaine 0.125% or 0.25% plain, bupivacaine 0.125% with adrenaline 1.25 µg/ml, bupivacaine 0.25% with adrenaline 2.5 µg/ml or the latter two solutions with added fentanyl 50 µg. Analgesia was unsatisfactory in 30% of the bupivacaine 0.125% groups without fentanyl. The addition of adrenaline, compared with bupivacaine 0.25% plain, gave faster onset and longer duration of analgesia ($p < 0.05$) which was similar to that found in both fentanyl groups. There were no differences in method of delivery or neonatal Apgar scores among groups. The least concentrated mixture that gave the best analgesia was the combination of bupivacaine 0.125% with adrenaline 1.25 µg/ml and fentanyl 50 µg.

Key words

*Anaesthesia; obstetric.
Analgesics; fentanyl.*

Epidural opioids have been used for obstetric analgesia since the discovery of opioid receptors in the brain and spinal cord,¹ but with limited success on their own.² Several groups demonstrated that the addition of opioids such as fentanyl to bupivacaine produced significantly longer duration and better quality of analgesia compared with either agent on its own.^{3,4} However, there were conflicting results at different concentrations of bupivacaine, and some studies used adrenaline-containing solutions. Adrenaline is often added to local anaesthetics in an effort to improve the duration and quality of analgesia.⁵ The addition of both fentanyl and adrenaline may further enhance the analgesic effects of the local anaesthetic through their combined effects at spinal opioid receptors and spinal alpha adrenoreceptors. Most previous studies compare only two or three study solutions and it is difficult to form an overall picture when different researchers have used different control groups, volumes of local anaesthetic, dose of fentanyl and adrenaline concentration. The aim of this study was to examine the effects of adding fentanyl and adrenaline to low concentrations of bupivacaine for epidural analgesia in labour.

Method

The study was approved by the Chinese University Ethics Committee, and informed consent was obtained from each patient. Ninety ASA 1 Chinese women with uncomplicated pregnancies of at least 37 weeks' gestation and single fetal cephalic presentation were studied in a double-blind manner. Patients were in established labour and had requested epidural analgesia before reaching 6-cm cervical dilatation. The women were assigned at random to receive one of the following study solutions prepared by diluting commercially available (Astra Pharmaceuticals) bupivacaine 0.5% or bupivacaine 0.5% and adrenaline 5 µg/ml (1:200 000) with physiological saline to a final volume of 10 ml: bupivacaine 0.125% plain; bupivacaine 0.125% with adrenaline 1.25 µg/ml; bupivacaine 0.125% with adrenaline 1.25 µg/ml and fentanyl 50 µg; bupivacaine 0.25% plain; bupivacaine 0.25% with adrenaline 2.5 µg/ml; bupivacaine 0.25% with adrenaline 2.5 µg/ml and fentanyl 50 µg.

The epidural technique was standardised and performed with the patient in the left lateral position. A 16-gauge Tuohy needle was used and the epidural space at L₂₋₃ or L₃₋₄

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Accepted 8 May 1990.

Table 1. First epidural dose, $n = 15$ in each group. The number of patients with unsatisfactory analgesia and median pain score values at 15 minutes, time to achieve lowest pain score, duration of analgesia and maximum percentage reduction in pain score. P, plain; A, adrenaline; F, fentanyl 50 μg . * $p < 0.05$, † $p < 0.01$ compared with corresponding P group; ‡ $p < 0.01$ compared with corresponding A group.

	Bupivacaine 0.125%			Bupivacaine 0.25%		
	P	A	FA	P	A	FA
Failure of analgesia	5	4	1	1	1	0
Pain score at 15 minutes	30	22	0	39	15	*0
Time to lowest pain score; minutes	28	22	15	38	15	*15
Analgesic duration; minutes	75	90	†‡120	90	*135	*120
Maximum % reduction in pain score	94	85	†‡100	100	100	100

identified with a loss of resistance to air technique. Three to five cm of catheter were left in the space. The study solution was administered slowly in divided doses after preloading the circulation with compound sodium lactate solution 500 ml.

Pain was assessed with a 10-cm visual analogue scale before the epidural was established. Pain scores were recorded after the first four uterine contractions and at 15-minute intervals after the study solution had been given. Maternal arterial blood pressure, heart rate and fetal heart rate were recorded at 5-minute intervals for 30 minutes. Failure of satisfactory epidural analgesia was defined as failure to achieve 50% reduction in initial pain score, or a request for further analgesia within 30 minutes of the first dose. Onset of analgesia was assessed by several indices: the time taken to achieve 50% reduction in initial pain score, the time to maximum analgesia and the pain score at 15 minutes. Quality of analgesia was calculated as the maximum percentage reduction in pain score. Duration of analgesia was taken as the time from administration of the first dose to the time when the patient requested further analgesia. The incidence of side effects such as nausea, vomiting, drowsiness, pruritus, shivering and urinary retention were recorded. Hypotension was defined as a systolic arterial pressure less than 100 mmHg.

Ten millilitres of the study solution was given when additional analgesia was requested, and if satisfactory then this solution was continued until delivery. Assessment of analgesia was repeated for the second epidural dose. The study solution was abandoned and bupivacaine 0.25% or 0.375% given instead, if pain relief was found to be inadequate after the second dose. The outcome of labour, neonatal birth weight and Apgar scores at 1 and 5 minutes were recorded.

Demographic and other parametric data were compared using one-way analysis of variance, while pain scores, analgesic duration and Apgar scores were compared using the Kruskal-Wallis test. Categorical data were compared using contingency table analysis with pooling of some groups. Differences were accepted as significant when $p \leq 0.05$.

Results

There were no demographic differences among the six groups. Overall, the mean (SD) age was 27.4 (4.5) years, height 156 (5.9) cm, weight 65.2 (8.0) kg and gestation 40 (1.3) weeks. There were no differences in parity among the groups with a total of 78 primiparae. Epidural analgesia was performed at a similar stage of labour among groups; the mean (SD) cervical dilatation was 2.7 (1.2) cm at that time. The course of labour appeared to be similar among groups with no differences in the number of induced labours or the number of cases that required augmentation with oxytocin.

There were differences in onset, quality and duration of analgesia among the groups. Analysis of results after the second epidural dose agree with findings from the initial dose (Tables 1 and 2).

Effect of concentration. There was a higher incidence of unsatisfactory analgesia in the bupivacaine 0.125% groups with and without adrenaline, compared with the bupivacaine 0.25% groups with and without adrenaline ($p < 0.05$). There were no differences between bupivacaine 0.125% with fentanyl and adrenaline, and bupivacaine 0.25% with fentanyl and adrenaline.

Effect of adrenaline. There were no differences between bupivacaine 0.125% and bupivacaine 0.125% with adrena-

Table 2. Second epidural dose. The number of patients with unsatisfactory analgesia and median values for time to achieve lowest pain score, and duration of analgesia. P, plain; A, adrenaline; F, fentanyl 50 μg . * $p < 0.05$ compared with corresponding P group; † $p < 0.05$ compared with corresponding A group.

	Bupivacaine 0.125%			Bupivacaine 0.25%		
	P	A	FA	P	A	FA
Number of patients	13	11	12	13	13	8
Failure of analgesia	2	0	0	1	0	0
Time to lowest pain score; minutes	45	30	15	30	10	15
Analgesic duration; minutes	90	90	*†165	75	105	*128

Table 3. Incidence of side effects and method of delivery with 15 patients in each group. P, plain; A, adrenaline; F, fentanyl 50 µg.

	Bupivacaine 0.125%			Bupivacaine 0.25%		
	P	A	FA	P	A	FA
Pruritus	0	0	2	0	0	2
Drowsiness	1	0	2	1	0	1
Nausea or vomiting	1	3	2	0	0	2
Shivering	0	4	3	4	2	4
Hypotension	3	0	3	4	0	1
Spontaneous vaginal	7	3	3	6	6	6
Ventouse or forceps	2	8	8	4	4	4
Caesarean section	6	4	4	5	5	5

line 1.25 µg/ml. Bupivacaine 0.25% with adrenaline 2.5 µg/ml gave longer duration and faster onset of analgesia than bupivacaine 0.25% plain ($p < 0.05$).

Effect of adrenaline and fentanyl. The addition of fentanyl 50 µg and adrenaline to bupivacaine 0.125% gave longer duration and better quality of analgesia compared with plain bupivacaine 0.125% or bupivacaine 0.125% with adrenaline ($p < 0.01$). Bupivacaine 0.25% with fentanyl 50 µg and adrenaline gave longer duration and more rapid onset of analgesia than plain bupivacaine 0.25% ($p < 0.05$). There were no differences between bupivacaine 0.25% with fentanyl and adrenaline and bupivacaine 0.25% with adrenaline alone.

There were no differences among the six groups when individual side effects were compared. However, the total number of side effects was greater in the groups that contained fentanyl ($p < 0.05$), particularly pruritus, which appeared only in the fentanyl groups (Table 3). The addition of fentanyl did not reduce the incidence of shivering. Urinary retention was difficult to assess since urinary catheterisation was routinely performed before all operative deliveries. Hypotension was rare and easily treated with intravenous fluid and left uterine displacement.

There was no difference in the outcome of labour among groups, with a total of 31 spontaneous vaginal deliveries, 30 instrumental deliveries and 29 Caesarean sections (Table 3). The median duration of the first and second stages of labour were 8.8 hours and 45 minutes respectively.

There were no differences among groups in fetal heart rate after epidural injection, Apgar scores at delivery or birth weight.

Discussion

Epidural opioids appeared to be ideal for pain relief in labour because they selectively affect pain perception while sparing the autonomic and motor pathways. Epidural morphine alone in clinical practice was relatively ineffective.⁶ However, epidural fentanyl 150–200 µg was effective only during the early part of labour,⁷ while other workers showed that epidural fentanyl 80–100 µg was effective for perineal analgesia.^{8,9} Nevertheless, the addition of fentanyl to low concentration bupivacaine has produced satisfactory analgesia for labour.¹⁰ The use of low concentrations of local anaesthetic may decrease the patient's motor block which in turn may prevent an increased incidence of operative deliveries.^{11,12} The reduced local anaesthetic requirement may decrease the incidence of potentially life

threatening complications from local anaesthetic toxicity and inadvertent intravascular or subarachnoid injection.

Adrenaline has been added to epidural local anaesthetic solutions to decrease systemic absorption of local anaesthetic and improve onset and prolong duration of analgesia. We preferred to dilute existing commercially available solutions than add adrenaline separately but this decreases the concentration of adrenaline with increasing dilution. We believe that this method is more practical and safer than adding adrenaline separately, although it was suggested that freshly added adrenaline provides a superior block.¹³

There are many factors which complicate the interpretation of results from studies of analgesia in labour. Patients are generally an homogeneous group, but their response to pain and the course of labour can be very different. Labours that are induced or augmented may produce more painful contractions.

One often forgotten factor is the change in pH of the resultant mixture when bupivacaine solutions are diluted. The pH of the study solutions ranged from 4.29 in the bupivacaine 0.25% with adrenaline mixture to 5.72 in the bupivacaine 0.125% plain mixture. An increase in pH may quicken the onset of analgesia but the effects of this have not been explored in the context of this and other similar studies.

We found that adrenaline 1.25 µg/ml was not effective in potentiating bupivacaine 0.125%, and 30% of patients had inadequate analgesia. Bleyaert *et al.*¹⁴ found that bupivacaine 0.125% with adrenaline 1.25 µg/ml gave satisfactory analgesia in more than 90% of their patients, but the total volume administered to each patient initially, including the test dose of the same solution, was at least 15 ml, while our patients received only 10 ml. The addition of adrenaline 2.5 µg/ml to bupivacaine 0.25% in our study caused faster onset and longer duration of analgesia compared with the plain solution. Similar results have been reported⁵ and the addition of adrenaline 3.33 µg/ml to bupivacaine 0.5% also reduced and delayed the incidence of hypotension.¹⁵ However, the addition of adrenaline to bupivacaine 0.5% during labour did not prolong analgesia and only slightly reduced systemic absorption of bupivacaine.¹⁶ Concern was expressed over the use of adrenaline in parturients because of possible adverse effects on uterine blood flow,¹⁷ but epidural adrenaline 26 µg did not cause any adverse neonatal effects.¹⁵

The addition of fentanyl 50 µg to bupivacaine 0.125% with adrenaline resulted in better and longer lasting analgesia than the plain or adrenaline-containing solution.

Bupivacaine 0.25% with adrenaline 2.5 µg/ml on its own provides good analgesia in most patients and it was difficult to demonstrate any significant benefit from the addition of fentanyl 50 µg. It was reported that the addition of both fentanyl and adrenaline to bupivacaine 0.25% provided longer duration of analgesia than the addition of either agent alone.¹⁸ We did not compare the fentanyl and bupivacaine groups with and without adrenaline in this study, but we have previously investigated the effects of fentanyl and bupivacaine mixtures in a study with similar methodology.¹⁹ The bupivacaine, fentanyl and adrenaline groups in that study had longer duration of analgesia than the corresponding groups without adrenaline.

Pruritus is a recognised side effect after epidural opioids, but this symptom was elicited only on direct questioning and the patients in this study thought itching was only mild. We did not show that epidural fentanyl decreased the incidence of shivering, but this has been reported previously.²⁰

Motor block was not evaluated and although the method of delivery was similar among groups, we cannot make any further conclusions, since some patients in the lower concentration groups received bupivacaine 0.25% or 0.375% after analgesia was assessed to be inadequate. Pain increases as labour progresses and it is not unusual to increase the frequency or dose of epidural solution. Our study groups are too small and not controlled sufficiently to correlate total epidural drug requirement with method of delivery.

There were no adverse neonatal effects in the fentanyl groups and fentanyl 50 µg appears to be a safe lumbar epidural dose to administer. Epidural fentanyl 150 to 200 µg gave mean maternal arterial fentanyl levels of 0.3 ng/ml and umbilical arterial fentanyl of 0.18 ng/ml without any respiratory depression to mother or neonate.⁷

Analgesia was best with the bupivacaine 0.25% with adrenaline 2.5 µg/ml and the two fentanyl mixtures. We see no advantage in using the more concentrated mixture since the two fentanyl groups were similar. We conclude that the combination of bupivacaine 0.125% with adrenaline 1.25 µg/ml and fentanyl 50 µg gives good pain relief and is a suitable choice for obstetric epidural analgesia.

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Predictability of difficult laryngoscopy in patients with long-term diabetes mellitus

E. REISSELL, R. ORKO, E-L. MAUNUKSELA AND L. LINDGREN

Summary

The laryngoscopic conditions of 62 diabetic patients who underwent renal transplantation or vitrectomy were studied. Anaesthesia was induced with fentanyl and a sleep dose of thiopentone. Conditions for direct laryngoscopy after 0.1 mg/kg vecuronium were scored from 0 to 3 (easy–very difficult). All patients gave their palm prints after operation which were scored: 0, phalangeal areas completely visible; 1, phalangeal areas partly visible; 2, phalangeal areas hardly visible; 3, only fingertips printed. The incidence of difficult laryngoscopy was 31%. The higher the scores in the palm test, the more difficult was the laryngoscopy. The correlation coefficient between these two factors was $r = 0.6$ ($p < 0.001$). Our study shows that joint rigidity possibly caused by tissue glycosylation may also involve laryngeal and cervical areas resulting in a strenuous laryngoscopy. A defective palm print is a warning sign for difficult laryngoscopy.

Key words

Intubation, tracheal; difficult. Complications; diabetes.

The management of diabetes mellitus has greatly improved during the last decades but complications may still require surgery; renal and pancreatic transplantations are performed on diabetic patients more frequently than 10 years ago. General anaesthesia with tracheal intubation is necessary for these operations. Salzarulo *et al.* in 1986 published the first report on difficult laryngoscopy in diabetic patients,¹ and since then there has been a growing interest in this problem. The incidence of difficult laryngoscopy was reported to be about 30% in patients with long-term type 1 diabetes mellitus.^{2,3}

Limited joint mobility syndrome (LJM) is present in 30–40% of type 1 diabetic patients,^{4,7} who are unable to approximate the palms of their hands, and their fingers will not bend backwards. This syndrome is associated with deficient growth, multiple joint contractures and thick, waxy skin. Glycosylation of tissue proteins from chronic hyperglycaemia seems to be responsible for this complication.^{8,9} This joint rigidity may also involve laryngeal and cervical areas resulting in a strenuous laryngoscopy. Limited joint mobility can be assessed with a palm test,¹⁰ which we evaluated, to predict laryngoscopy conditions in patients with type 1 diabetes mellitus.

Patients and methods

Sixty-two patients with type 1 diabetes mellitus were studied. Forty-two underwent renal transplantation in the Department of Surgery and 20 were operated on for vitrectomy in the Department of Ophthalmology. The study was approved by the Ethics Committees and informed consent was obtained from all patients.

Premedication was oral diazepam 0.15 mg/kg and intramuscular oxycodone, 0.14 mg/kg. Anaesthesia was induced with fentanyl 2.4 (SD 0.8) $\mu\text{g/kg}$ and a sleep dose of thiopentone (4.0 (1.1) mg/kg). Adequate muscle relaxation was achieved with vecuronium 0.1 mg/kg. The lungs were ventilated with 100% oxygen, after the administration of vecuronium, until no response was obtained to the train-of-four. Conditions for direct laryngoscopy were judged by the anaesthesiologist who attended the case. Laryngoscopy was evaluated as easy (0) when direct visualisation of any part of the vocal cords was possible. Conditions were judged relatively easy (1) when laryngoscopy was hampered by a stiff neck but the larynx could be visualised. Conditions were relatively difficult (2) when the larynx could not be visualised because of a stiff neck, limited mouth opening

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Accepted 4 April 1990.

or a hanging epiglottis. Laryngoscopy was deemed difficult (3) when there was a hanging epiglottis and the anaesthetist needed several attempts to accomplish a successful tracheal intubation with the aid of an introducer.

The palm of the dominant hand was painted with black ink using a roller 2 days later. All patients gave their palm prints with fingers spread on white paper. A person unaware of the laryngoscopy conditions scored the prints.

Palm prints were scored 0 (Fig. 1a) when all phalangeal areas were visible, but 1 (Fig. 1b) when phalangeal areas were only partly visible. Special attention was paid on the metacarpophalangeal and proximal interphalangeal joints of the fifth and fourth fingers. If the phalangeal areas were barely visible i.e. the changes had spread into third and second interphalangeal joints, the palm print was scored 2 (Fig. 1c); the palm print was 3 when only fingertips were printed (Fig. 1d).

A regression analysis was drawn between the scores of the palm prints and laryngoscopy conditions. Parametric data were analysed with analysis of variance. The results are expressed as mean (SD).

Results

Table 1 shows the demographic data for all patients. The incidence of microvascular complications and neuropathy in both groups is presented in Table 2. The incidence of difficult laryngoscopy (scores 2 or 3) was 31%. The higher the scores were in the palm prints the more difficult was the laryngoscopy. The correlation coefficient between these two factors was $r = 0.6$ ($p < 0.001$) (Fig. 2). In 60% of all 62 cases the palm print score was exactly the same as the laryngoscopy conditions score. Laryngoscopy was found very difficult in 16% of all cases and required the use of an introducer. The mean glycosylated haemoglobin in this group was 9.9 (1.5) and for the others 9.6 (1.6) (ns). In one case, the history of a very difficult blind intubation before renal transplantation some years before, indicated awake

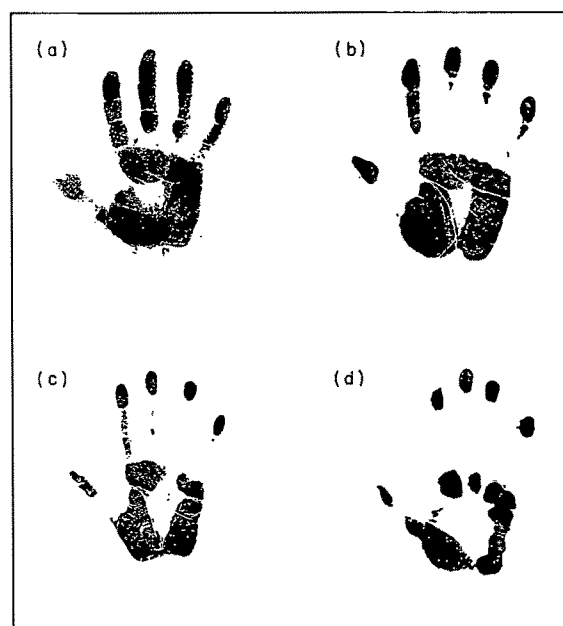


Fig. 1(a). A complete palm print (score, 0) in a patient with easy laryngoscopy conditions (score, 0). **(b).** A palm print (score, 1) deficient in the interphalangeal areas of the fifth and fourth fingers. Laryngoscopy conditions were scored 1 in this patient. **(c).** A palm print (score, 2) where changes have spread into the interphalangeal areas of the third and second fingers. Laryngoscopy was found difficult (score, 2). **(d).** A palm print with only fingertips printed (score, 3). Several attempts were needed and the introducer was used for laryngoscopy (score, 3).

fibreoptic laryngoscopy. This patient's laryngoscopy conditions score as well as his palm print were judged as very difficult or 3. The prayer sign of a 31-year-old woman with a palm print scored 3 (Fig. 1d) is seen in Figure 3. Laryngoscopy in her case was also extremely difficult. Two cases were operated on for Dupuytren's contracture.

Table 1. Demographic data for 62 diabetic patients, mean (SD).

	Patients for renal transplantation (<i>n</i> = 42)	Patients for vitrectomy (<i>n</i> = 20)
Males/females	24/18	5/15
Age; years	39.1 (9)	39.8 (13)
Weight; kg	63.3 (10)	70.8 (14)
Height; cm	167.5 (10)	171.9 (12)
Duration of diabetes; years	24.7 (6)	24.5 (6)
HbA _{1c}	9.6 (2)	9.1 (1)

Table 2. Complications of diabetes and need for dialysis treatment.

	Renal transplantation	Vitrectomy
Number of patients	42	20
Dialysis	42	2
Retinopathy	42	20
Neuropathy	13	6

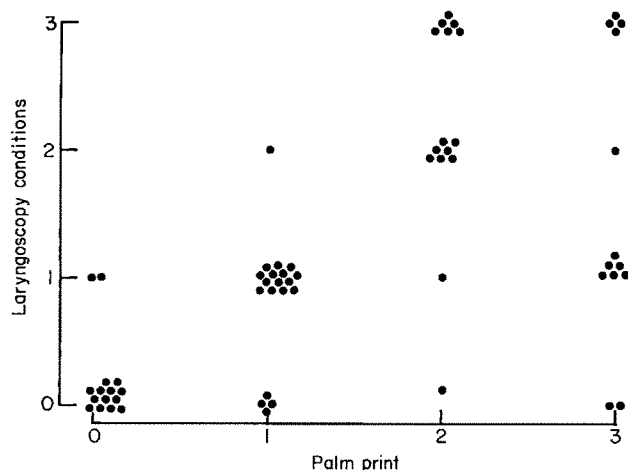


Fig. 2. Relationship between palm print scores and laryngoscopy conditions scores. A regression analysis gave an equation $y = 0.58 + 0.32x$ with $r = 0.6$ ($p < 0.001$). See Note added in proof.

Discussion

A high incidence of difficulties during tracheal intubation was observed in patients with long-term diabetes mellitus.¹¹ Earlier studies showed that this complication is met in approximately 30% of all diabetic patients, while the incidence of difficult laryngoscopy is 3% in nondiabetic patients.^{2,3} The incidence of difficult laryngoscopy in our study was 31% in type 1 diabetic patients. The palm print scores correlated well to the laryngoscopy conditions scores, and mean glycosylated haemoglobin had no prognostic value for difficult laryngoscopy.

Two case reports on unanticipated difficulties with laryn-

goscopy in diabetes mellitus have been published previously. In one report a candidate for renal transplantation was described to have the 'stiff joint syndrome'.¹ This patient had rapidly progressive microangiopathy, nonfamilial short stature, tight waxy skin and limited joint mobility. Inability to extend the atlanto-occipital joint detected radiologically was thought to be the cause of these difficulties. Bedside examination of the neck before operation was misleading. Orko *et al.*¹² found difficulties in tracheal intubation in six out of 14 diabetic patients. The major reason for difficulties in this study was stiffness of the neck, but mouth opening and external jaw and facial appearance were normal. An experienced anaesthetist needed 22 minutes to accomplish a successful intubation in the most difficult case.

Limited joint mobility (LJM) syndrome described in 1957¹³ is present in 30–40% of insulin-dependent diabetics.^{4–7} Patients with LJM are unable to approximate the palms of their hands, and their fingers will not bend backwards. Changes usually begin in the metacarpophalangeal and proximal interphalangeal joints of the fifth finger and spread medially. Thick, waxy scleroderma-like skin can be observed at this stage. Ultimately all joints may be affected, including fingers and hands, wrists, elbows and other large joints, and the cervical and thoracolumbar spine.¹⁴ LJM is associated with the microvascular complications of diabetes i.e. retinopathy and nephropathy. In Rosenbloom's series 83% of patients with moderate or severe LJM had retinopathy.⁷

Many complications of diabetes mellitus are related to glycosylation of tissue proteins associated with chronic hyperglycemia. Prevailing evidence suggests that LJM may be another example of tissue glycosylation. Diabetic patients may have an abnormality of collagen metabolism⁸ and increased cross-link formation.⁹ Collagen fibrils are abnormally stable in diabetic patients but these changes are potentially reversible.^{15,16} We propose that difficulties at laryngoscopy are from tissue glycosylation in laryngeal and cervical areas.

LJM has often been unnoticed at the time of proposed operation because it is usually a painless disorder causing minimal disability. Bedside diagnosis is relatively easy. The patient's hands should be observed for thick, waxy skin; hands should be placed in the 'prayer' position and the inability to oppose interphalangeal joints of fingers can be assessed. The incomplete flexion and extension of wrists and elbows should be observed as well as mobility of spine and lateral bending of neck. These measures can easily be included in the routine pre-operative evaluation of diabetic patients. Assessment of the motion at the temporomandibular and cervical vertebral joints can be misleading. An objective measure of LJM can be obtained by painting the fingers and assessing the so-called palm print.¹⁰ Other studies have shown that flexion-extension radiography of the cervical spine may be warranted.¹⁷

Diabetic gastric motor dysfunction, residual gastric contents and hyperacidity¹⁸ increase the risk of the diabetic patient during induction of anaesthesia. Correct airway assessment is therefore of great importance. The presence of LJM should be noted as a warning sign of difficult laryngoscopy in type 1 diabetic patients and the anaesthesiologist in charge of the case can be prepared for difficulties and fiberoptic or awake tracheal intubation can be used when necessary.

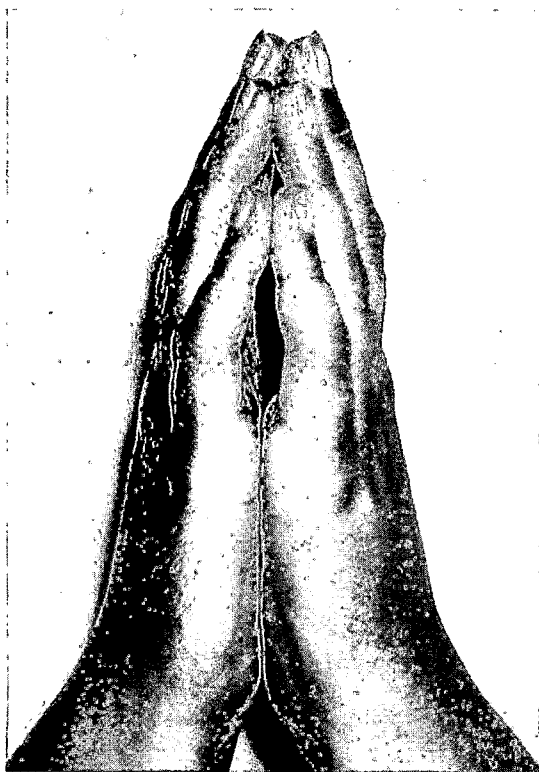


Fig. 3. The hands of a 31-year-old diabetic woman in a prayer sign. Her palm print is shown in Fig. 1d. Conditions for laryngoscopy were very difficult (score, 3).

Acknowledgments

The authors are indebted to Dr P. Reissell for discussions and criticism. This work was supported by a grant from the Paulo Foundation, Helsinki, Finland.

Note added in proof

The linear regression analysis may not be the most appropriate procedure. The Mantel extension test¹ to the Chi-square test ($\chi^2 = 21.5$, $p < 0.001$) and Spearman rank correlation coefficient (0.59) confirms the significance of our data.

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A randomised double-blind study of interpleural analgesia after cholecystectomy

A. LEE, D. BOON, P. BAGSHAW AND P. KEMPTHORNE

Summary

Continuous interpleural analgesia provided by 4 hourly injections of 20 ml bupivacaine 0.5% with adrenaline 5 µg/ml was compared with placebo in a randomised, double-blind study after cholecystectomy. All patients self-administered intravenous morphine using a patient-controlled analgesia device. There was a highly significant difference in mean morphine consumption between the groups (72 mg as compared with 22 mg). Visual analogue pain scores tended to be lower in the bupivacaine group throughout and this was significant at 2 hours. Respiratory function measurements were not significantly different between the groups. The mean peak venous plasma bupivacaine concentration after the sixth dose was 3.03 µg/ml and no symptoms suggestive of local anaesthetic toxicity occurred. It is concluded that this regimen can provide effective and continuous analgesia after cholecystectomy and that combined administration of interpleural bupivacaine and systemic morphine is more effective than morphine alone in the immediate postoperative period. The doses of bupivacaine required for optimal use of the technique lead to significant total plasma bupivacaine concentrations within 24 hours.

Key words

*Anaesthetic techniques, regional; interpleural.
Pain; postoperative.*

Pain relief after unilateral thoraco-abdominal operations may be provided by the administration of bupivacaine into the pleural space.^{1–3} A duration up to 27 hours after a single injection of 0.5% bupivacaine 20 ml with adrenaline 5 µg/ml has been reported,¹ but recent studies suggest that analgesia is usually shorter-lived.^{4,5} In our experience, analgesia invariably wears off within 5 hours of administration of the above dose, although 4 hours of pain relief is consistently achieved. Patients who require little or no opioid supplementation may experience extremely painful episodes because the effect of the local anaesthetic wears off rapidly and there is no background analgesia. This intermittent absence of pain relief tends to exaggerate the efficacy of the technique and the possible beneficial effects on respiratory function.^{2,6} It needs to be shown that interpleural analgesia has advantages compared to current regimens before it is adopted for widespread clinical use. The plasma concentrations of bupivacaine attained when interpleural block is maintained using repeated bolus injections of local anaesthetic for prolonged periods remain to be determined.

The aims of this study were to compare interpleural analgesia to placebo in a randomised, double-blind manner and assess whether a combined interpleural and systemic opioid technique provides better pain relief than opioid alone. Local anaesthetic blockade was maintained, and episodes of severe pain were avoided. We examined the effects of interpleural analgesia on respiratory function and measured the plasma concentrations of bupivacaine that may be expected when an intermittent bolus technique is used in an optimal fashion.

Methods

Twenty patients (aged 24–70 years, ASA 1 or 2), undergoing cholecystectomy were admitted to the study, which was approved by the local ethics committee. All patients received instruction before operation in the use of the Cardiff Palliator and visual analogue scales for scoring pain. Baseline measurements of FEV₁, FVC and the FEV₁/FVC ratio were made with the patient in the sitting position using a wedge spirometer (Vitalograph).

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Accepted 31 May 1990.

Triazolam 0.25 mg was given orally one hour before surgery, and anaesthesia was induced with thiopentone and maintained with nitrous oxide 70% and isoflurane 0.5% in oxygen. Alfentanil was administered intravenously at induction, followed by an alfentanil infusion at 1 ($\mu\text{g}/\text{kg}$)/minute; the patients were paralysed with vecuronium and ventilation was controlled. The surgeon did not cross the midline with the skin incision. The alfentanil infusion rate was reduced to 0.5 ($\mu\text{g}/\text{kg}$)/minute 10 minutes before completion of surgery, and after surgery, with the patient still paralysed and anaesthetised, an 18-gauge Tuohy needle was used to insert an extradural catheter interpleurally through the eighth intercostal space, in the posterior axillary line, on the right side. This procedure was performed with the patient in the left lateral position using an air-filled syringe and a loss of resistance technique. The breathing system was disconnected immediately before puncturing the parietal pleura, and the ability to thread 20 cm of catheter rapidly was taken as confirmation that the catheter was not sited superficially to the parietal pleura. All anaesthetic agents were discontinued after withdrawing the needle, and residual paralysis was reversed with neostigmine.

The patients were randomly allocated to two groups by the hospital pharmacy who provided coded ampoules for interpleural injection. The patients in one group received six doses, each of 20 ml 0.9% saline at 4-hourly intervals through the interpleural catheter. The patients in the other group received six doses, each of 20 ml 0.5% bupivacaine with adrenaline 5 $\mu\text{g}/\text{ml}$ at 4-hourly intervals. All interpleural injections were made by an anaesthetist (A.L., D.B.) with the patient supine. The upper half of the patient's body was placed in a 30° head-up position one minute after injection. The first interpleural injection was made immediately after catheter insertion with the patient supine on the operating table. The sixth injection was made at 20 hours.

All patients were allowed to self-administer 2 mg increments of morphine intravenously after operation using a Cardiff Palliator with a lockout interval of 5 minutes. All patients at 24 hours, after completion of the study period, received a seventh interpleural injection of 20 ml 0.5% bupivacaine with adrenaline 5 $\mu\text{g}/\text{ml}$. The placement of the catheter was then confirmed by demonstrating loss of cold sensation in several thoracic dermatomes on the right side using an ice cube.

Assessments were made by the attending anaesthetist (A.L., D.B.) at 2, 4, 6, 8, 12 and 20 hours after operation using 10-cm linear visual analogue scales (no pain—the worst pain I can imagine). Respiratory function tests were repeated at 4 and 24 hours after operation with the patient in the sitting position. Venous blood samples were taken before and at 5-minute intervals for 30 minutes after the sixth interpleural injection, and these were stored at

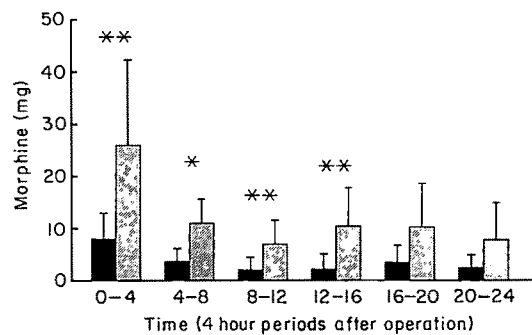


Fig. 1. Morphine consumption during the first 24 hours after surgery ($p < 0.01$ significant). ■, bupivacaine; ▨, saline; * $p < 0.001$; ** $p < 0.01$.

−20°C. The samples from patients who had received placebo were discarded on completion of the study, and samples from patients who received bupivacaine were analysed for plasma bupivacaine concentrations. Any side effects that occurred during the study were recorded.

Comparisons were made between groups using Student's t or Mann-Whitney tests with Bonferroni corrections for multiple testing where appropriate.

Results

There were no significant differences between the groups in age, height, weight, sex and smoking habits (Table 1). FEV₁, FVC and FEV₁/FVC ratio were within the normal range based on standard tables for all patients, and the groups were comparable.

The mean dose of morphine consumed in the first 24 hours after operation was 21.6 mg in the bupivacaine group and 72.4 mg in the saline group ($p < 0.001$). Significantly more morphine was consumed in the saline group in every 4-hour time period up to 16 hours (Fig. 1). Visual analogue scores tended to be higher in the saline group throughout the study (Fig. 2), and this was significant at 2 hours ($p < 0.01$).

There was a highly significant decrease in FEV₁ and FVC at 4 hours after operation in both groups and this decrease was maintained at 24 hours (Table 2). There were no significant differences in respiratory function tests between the groups at any time.

The mean peak venous plasma bupivacaine concentration after the sixth dose was 3.03 $\mu\text{g}/\text{ml}$ and occurred between 10 and 30 minutes after injection. The highest

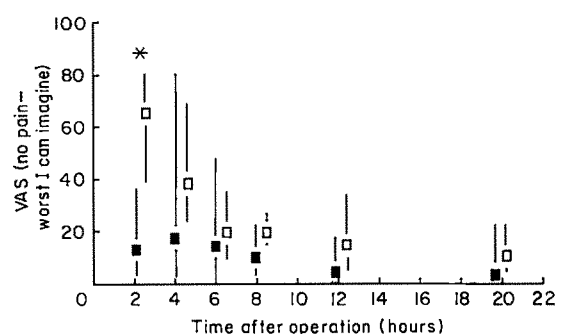


Fig. 2. Visual analogue pain assessments (no pain—the worst pain I can imagine) after surgery. —■—, bupivacaine (median/quart); —□—, saline (median/quart); (* $p < 0.01$ significant).

Table 1. Demographic data, mean (SD).

	Age (years)	Height (m)	Weight (kg)	Sex (M/F)	Smokers (Y/N)
Bupivacaine group	50.3 (15.1)	1.65 (0.08)	72.1 (12.2)	3/7	3/7
Saline group	50.7 (14.5)	1.69 (0.10)	77.7 (14.6)	4/6	3/7

Table 2. Respiratory function tests, mean (SD).

	Bupivacaine group (ml)	Saline group (ml)
<i>Pre-operative values</i>		
Predicted FEV ₁	2778 (669)	2924 (567)
Measured FEV ₁	3037 (997)	3104 (723)
Predicted FVC	3672 (804)	3920 (761)
Measured FVC	3695 (1192)	3839 (981)
<i>4 hours after operation</i>		
FEV ₁	1415 (461)	1697 (467)
FVC	1739 (682)	2174 (513)
<i>24 hours after operation</i>		
FEV ₁	1721 (683)	1704 (450)
FVC	2112 (802)	2089 (573)

bupivacaine concentration measured in any patient was 3.91 µg/ml (Fig. 3). No patient had any symptoms suggestive of local anaesthetic toxicity at any time elicited by spontaneous reporting or repeated direct questioning.

Discussion

This study confirmed the efficacy of interpleural bupivacaine after cholecystectomy and shows that continuous pain relief can be achieved using an intermittent bolus technique of 20 ml 0.5% bupivacaine with adrenaline 5 µg/ml every 4 hours. Combined administration of interpleural bupivacaine and systemic morphine is more effective than morphine alone in the first few hours after surgery. Previous studies in postoperative patients with no analgesia have demonstrated a significant decrease in visual analogue pain score after the interpleural administration of local anaesthetic.^{2,6} This confirms the pain relieving effect of interpleural local anaesthetic, but does not show that it is as effective or better than other methods of analgesia. A controlled, double-blind comparison with established analgesic regimens on the day of surgery has not previously been conducted. Studies performed on the day of operation have either been unblinded or have used a local anaesthetic infusion technique.^{5,7}

Continuous infusions may be effective after thoracotomy in children, but appear to be of less use after thoracotomy or cholecystectomy in adults.⁷⁻⁹ This may be related to the distribution of local anaesthetic in the pleural space. Large bolus doses of solution are retained in the pleural cavity for

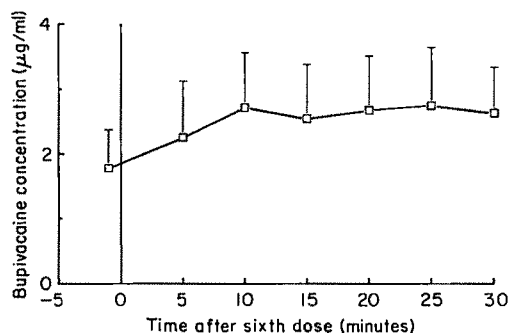


Fig. 3. Mean venous plasma bupivacaine concentrations before and after the sixth dose of interpleural bupivacaine (—□—, mean + SD).

appreciable periods of time and spread along the dorsal aspect of the cavity from diaphragm to apex.² There may be rapid flow of solution to the mediastinal aspect of the pleural cavity with continuous infusion of drug because of the pressure gradients that exist within the cavity.¹⁰ The results from continuous blocks achieved by intermittent bolus techniques may well differ from the effects obtained by infusion of similar doses.

The effect of interpleural blockade on respiratory function after upper abdominal surgery is controversial.^{2,4,7,11} Some studies have examined the effect of interpleural local anaesthetic in patients with no analgesia at all after an upper abdominal operation and shown that there is a significant improvement in respiratory function.² This does not exclude the possibility that interpleural blockade may have detrimental effects on diaphragmatic function, nor does it show that interpleural analgesia is preferable to systemic opioids. Poorer respiratory function in the presence of interpleural blockade compared to systemic opioids alone has been demonstrated.¹¹ However, in that study the patients receiving systemic opioids had better respiratory function than has usually been described after upper abdominal surgery and this may represent a chance finding. We were unable to demonstrate any difference in overall function in the present study compared to patient-controlled opioid analgesia. More detailed study of respiratory function in the presence of interpleural blockade is indicated, particularly in relation to diaphragmatic function which may be impaired.

The venous plasma concentrations of bupivacaine after the sixth dose approached previously reported toxic levels,¹² and were higher than the plasma concentrations reported after one or two doses of interpleural bupivacaine.^{2,6,13,14} Symptoms suggestive of local anaesthetic toxicity were specifically sought and were not present at any time. The lack of toxic effects at these plasma concentrations is related to the marked increase in plasma alpha₁-acid glycoprotein known to occur after surgery.¹⁵ This protein provides the main binding sites for bupivacaine and with an increased availability of binding sites the total plasma bupivacaine concentration may increase without an increase in free drug concentration. Similar plasma concentrations have been reported during extradural infusions, again without clinical evidence of systemic toxicity.¹⁶ The rate of rise of plasma concentration is important and the gradual increase that occurs in the clinical setting compared to the acute rise in volunteer studies helps to account for the lack of systemic effects.¹⁷ Continuous administration of bupivacaine at these doses for periods exceeding 48 hours should be viewed with some caution. Increases in the total plasma bupivacaine concentration to values exceeding 5 µg/ml have been measured during post-operative extradural infusions that have been in progress for over 2 days.¹⁸ No clinical evidence of bupivacaine toxicity was reported, but these patients had relatively stable and low plasma concentrations of bupivacaine for the first 48 hours. Measurement of the free drug concentration was not performed during these late increases in total concentration, but would have been of value.

The necessity for frequent top-ups limits the use of intermittent interpleural techniques, and studies conducted in a double-blind manner have confirmed that the duration expected from a single bolus is around 4 hours.^{2,4,19} Further investigation of interpleural local anaesthetic infusions is

desirable, but they may not prove to be as useful as intermittent bolus techniques.

It is concluded that continuous, effective analgesia can be maintained by regular interpleural injections of bupivacaine after cholecystectomy and that analgesia superior to that obtainable from systemic opioids alone is possible. Significant total plasma bupivacaine concentrations are present after 24 hours.

Acknowledgment

We should like to thank Astra Pharmaceutical, New Zealand for measurement of plasma bupivacaine concentrations.

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Anaphylactic anaesthetic reactions

The value of paper radioallergosorbent tests for IgE antibodies to muscle relaxants and thiopentone

E. S. K. ASSEM

Summary

The three currently available paper radioallergosorbent tests ('suxamethonium', alcuronium and thiopentone) were evaluated. 'Suxamethonium' radioallergosorbent test (which employs choline conjugated to paper discs) proved to be reliable in the detection of allergy to neuromuscular blockers, which were confirmed as the most common cause of anaphylactic reaction during general anaesthesia. Thiopentone radioallergosorbent test may also be useful, and is recommended in conjunction with 'suxamethonium' radioallergosorbent test in the preliminary investigation of reactions. Patients with positive 'suxamethonium' radioallergosorbent test usually require further testing, including alcuronium radioallergosorbent test, skin testing with a wide range of drug concentrations or leucocyte histamine release test.

Key words

Neuromuscular relaxants; suxamethonium, alcuronium.
Allergy; anaphylaxis, thiopentone.

Much controversy surrounds anaesthetic allergies. This debate was highlighted by recent medicolegal arguments that followed fatal cases of suxamethonium anaphylaxis.^{1–10} Anaphylactic reactions are rare according to most surveys^{11–13} and occur in the order of 1/4500–20 000 general anaesthetics, i.e. 175–778 cases per year in UK. Some sources estimate an incidence of 1/600 and > 5000 cases annually in UK,^{14–16} but this is unlikely.⁵ These reactions are not thought to contribute significantly to anaesthetic mortality, but previous studies^{17,18} did not seek specific information about them,⁶ and allergy tests were not performed.^{1,6,17,18} Simple tests, such as the ones described herein, were not available. Adverse reactions to drugs are under-reported^{19–20} and the diagnosis is frequently missed²¹ because unexpected effects such as cardiac arrhythmia, depression of contractility and cardiac arrest may be the presenting manifestations.^{8,22,23} Therefore, there is a definite need for simple but reliable tests for anaesthetic reactions.

A 20-year survey of English and French language literature showed that the so-called 'anaphylactoid' reactions are associated most commonly with the administration of neuromuscular blockers (NMB) and, less frequently, intravenous anaesthetics.²⁴ The reactions occur usually during induction of anaesthesia. Use of the term 'anaphylactoid' indicates some degree of uncertainty about the mechanism.^{25–30} Nevertheless, there is substantial evidence that both the clinical immediate reactions to NMB and the

NMB-induced histamine release from the basophil leucocytes (HRL, *in vitro*) of affected patients are IgE-mediated.^{8,30–33} The HRL test,^{28–30} despite some clear advantages, is too elaborate, time-consuming and expensive to be used routinely for diagnosis and 'screening'; hence the potential value of radioallergosorbent tests (RASTs). These tests measure allergen-specific IgE antibody in serum (significant levels of which indicate immediate-type allergy), using a conjugate of the allergen to a 'solid phase' (paper discs here) to bind (absorb, hence an allergosorbent test) the antibody, and radioisotope (or enzyme)-labelled anti-IgE to measure allergen disc-bound IgE antibody. This measurement is directly proportional to the serum concentration of allergen-specific IgE. The value of currently available RASTs and their place among other tests are discussed.

Patients and methods

Since 1972, 105 patients with 'hypersensitivity' reactions during general anaesthesia (GA) were referred to this centre and 79 of them were investigated. Of the 79 patients (57 females and 22 males aged 7–57 years) 71 had severe systemic anaphylaxis, one of which resulted in a fatal outcome.

Details of seven patients were published as case reports.^{8,34–37} A summary of clinical manifestations in the 79

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Accepted 8 May 1990.

Table 1. Summary of clinical manifestations of allergic reaction in the 79 patients who have been investigated.

Manifestation or complication	Number of patients in whom it was recorded
Death*	1
Reaction on two occasions	8
Blood pressure	
Low	71
Unrecordable	17
Heart: rate and rhythm	
Sinus tachycardia	65
Cardiac arrest	7
Arrhythmia	8
Fast ectopic	3
Slow	3
Very slow	2
Cardiac ischaemia*	1
Severe bronchospasm + hypotension	15
Bronchospasm only	5
Brain damage due to cardiac arrest	4
Extensive*	1
Moderately severe but largely reversible	1
Mild with complete recovery	1
Mild but with residual deficit (memory)	1
Skin manifestations only	3
Miscellaneous complications	
fetal loss (Caesarean section, total 4)	1†

*One case. †Misdiagnosis, pulmonary embolism led to massive vaginal bleed.

patients who were investigated is shown in Table 1. Forty normal controls were investigated; 28 of them had had GA, and none suffered a reaction. The 79 patients and 40 controls were investigated according to the summary shown in Table 2.

Skin tests. Prick (through a drop of allergen solution, 1–100 mm) and intradermal (i.d., approximately 0.025 ml, different concentrations, 1–1000 µM) tests were carried out as described previously.^{34,35}

Measurement of histamine release from leucocytes (HRL) was performed as described previously.³⁴ Briefly, leucocytes were separated from heparinised blood samples, washed three times with buffer, incubated with 'allergen' for 30 minutes at 37°C, spun down and the supernatant removed. Histamine in the supernatant (released histamine) and in the cell pellet (residual histamine) were measured (after addition of perchloric acid, final concentration 0.4 M, and precipitation of protein by spinning) by an automated spectrofluorometric procedure (Technicon). Histamine release (into supernatant) was expressed as per cent of total (supernatant + residual). HRL inhibition studies were carried out by pre-incubation of leucocytes, at 4°C and in calcium-free buffer, with choline, alcuronium and other compounds containing a quaternary ammonium group, prior to the normal procedure. Repeat HRL was carried out in many patients for confirmation, study of crossreac-

tion and finding a safe alternative, and for follow-up (up to 17 years). Paper RASTs were developed in conjunction with Pharmacia Ltd and testing (carried out in triplicate) was started in 1987.

Allergen discs. There was one disc for thiopentone (THIO) and two for the neuromuscular blockers (NMB), consisting of either choline (CHOL, which contains one quaternary ammonium group, QAG) or alcuronium (ALC, with two QAG, Fig. 1), coupled covalently to paper discs via a divinyl sulphone spacer arm.

Control discs. These were conjugates of human serum albumin (HSA) to paper.

IgE antibodies. Allergen discs were incubated with patients' sera at room temperature for 3 hours, followed by washing three times with HEPES (N-2-hydroxyethylpiperazine-N-2-ethane sulphonic acid)-buffered saline (HBS) and then the addition of ¹²⁵I-labelled anti-human IgE (50 µl, containing approximately 0.2 pM pure anti-IgE) and incubation at room temperature overnight. The discs were then washed three times with HBS and counted in a gamma counter. Results (disc uptake of labelled anti-IgE that is directly proportional to the concentration of serum allergen-specific IgE antibody) were expressed as uptake of ¹²⁵I-labelled anti-IgE, as % of total amount added (mean of the triplicate samples).

IgG antibodies. These were detected by a similar technique, using 0.2 pM ¹²⁵I-labelled sheep anti-human IgG (Amersham International).

RAST-inhibition studies. Serum was pre-incubated with different concentrations of choline, alcuronium, vecuronium and decamethonium, before performing RAST, and the effect of such treatment was compared with that of buffer. RAST elution studies were carried out with the same compounds, in an attempt to elute IgE antibodies bound to allergen discs, after RAST.

Table 2. Investigation of the 79 patients and 40 controls.

	Patients	Controls
HRL	79	20
RAST	44	40
Skin tests	60	26

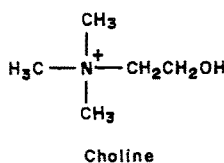


Fig. 1. Chemical structure of choline and some of its relevant derivatives or related compounds. All contain a quaternary ammonium antigenic (haptenic) group(s); acetylcholine, suxamethonium (succinylcholine) the depolarising neuromuscular blocker (NMB), and alcuronium the 'competitive' NMB. The first and last of those compounds were covalently linked to paper discs in order to detect sensitising (IgE) and non-sensitising (IgG) antibodies to the common and simple quaternary ammonium group (QAG) in choline derivatives and to the more specific and complex example of OAG in alcuronium.

Timing of blood samples. The first one was collected between 2 days and 4 months after the reaction (same for HRL). Further samples were collected at follow-up (up to 17 years,³⁵ Appendix) that took place at different intervals. Sera were stored at -20 to -60°C until tested, but many were tested while fresh.

Results

Histamine release from leucocytes (HRL)

No release could be elicited in the 20 normal patients tested (net release, after subtraction of spontaneous release in aliquots incubated with buffer alone, 0%), either with

NMB or thiopentone. The overall results of HRL in the patient group are summarised in Table 3. HRL was positive with NMB in 51 out of the 71 patients with systemic anaphylaxis (46 females and five males). Net HRL with NMB reached up to 75%. Three of those 51 patients also had a positive (but weaker) HRL with thiopentone, and another three had a weak response to premedicants (opioids and anticholinergics). Three patients gave a positive HRL with thiopentone only. In all patients with a positive HRL with thiopentone, histamine release was relatively small (only up to 20%). None of the eight patients with less severe nonsystemic reaction gave a positive HRL. Two examples of HRL with NMB are shown in Figure 2. Details of HRL and its comparison with the RAST will be

Table 3. Summary of patients reported and investigated in the period 1972–1989, and result of the histamine release from leucocytes (HRL).

Period	1972-76	1977-81	1982-86	1987-89	Total
Number reported	65	13	13	14	105
Number investigated by HRL	45	9	11	14	79
<i>Severe systemic reactions</i>	42	9	9	11	71
Number positive	42/42	9/9	9/9	11/11	71
<i>Relaxants/anaesthetics (induction)</i>					
Relaxant(s) only	19	9	8	9	45
Thiopentone only	3				3
Relaxant + weak thiopentone	1		1	1	3
Alphaxalone-alphadolone	14				14
Propanidid	3				3
<i>Premedicants (all weak + strong with relaxant)</i>					
Opioid	1				1
Anticholinergic	1				1
Opioid + anticholinergic				1	1
<i>Positive with relaxant only or mainly</i>					51
<i>Positive with relaxant and (or) thiopentone</i>					54
<i>Less severe 'nonsystemic'</i>	3	0	2	3	8
Number with positive HRL	0	0	0	0	0

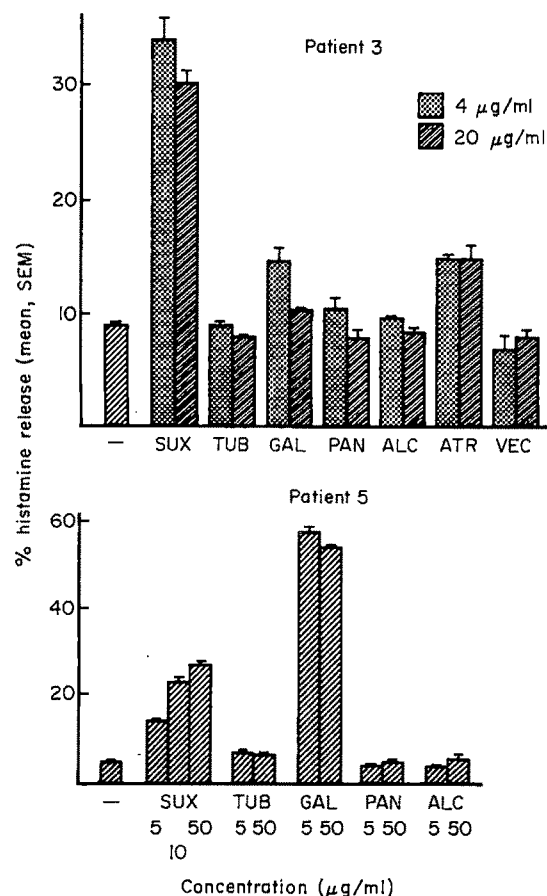


Fig. 2. Leucocyte histamine release test showing the extent of crossreaction to the different neuromuscular blockers (NMB) in two examples from the 36 patients with allergy to NMB. *Top:* patient 3, who was seen in 1987, had had an anaphylactic reaction after repeat exposure to suxamethonium (SUX). The test was carried out with the seven NMBs current in UK; SUX, tubocurarine (TUB), gallamine (GAL), pancuronium (PAN), alcuronium (ALC), atracurium (ATR) and vecuronium (VEC). *Bottom:* a patient who was first seen in 1981; she had had a reaction during her first general anaesthetic, which included SUX. Note the release of histamine with SUX and even greater release with GAL (–, control).

published elsewhere. HRL was positive with alphaxalone–alphadolone in 14 patients, and with propanidid in three (all in the early part of the study, both drugs withdrawn in 1983/84).

Neuromuscular relaxant RAST

IgE RAST. Incubation of allergen discs with serum, followed by their incubation with 125 I-labelled anti-IgE, washing and counting the radioactivity remaining on them ('uptake' expressed as % of total added) gives a measure of allergen-specific IgE antibodies in the serum. In the control group (normal patients) mean uptake and standard deviation by CHOL discs were 0.67 and 0.13% respectively. In ALC RAST in normal patients mean and SD were 1.0 and 0.79% respectively. A RAST test was considered positive if the measurement was above mean + 3 SD of the normal (i.e. 1.06% for CHOL and 3.37% for ALC, a cut-off that almost excludes every normal patient). Of the 35 patients with a systemic anaphylactic reaction (and positive HRL, excluding the case of reaction to thiopentone only), 33

showed a positive NMB RAST; 17 with CHOL discs only, 15 with both CHOL and ALC discs and one with ALC only. In addition, one of the eight patients, with less severe (nonsystemic, all with negative HRL) reaction was also RAST positive. Anti-IgE uptakes of up to 26.4% and 13.4 by CHOL and ALC discs respectively were obtained in the patient group. The results are summarised in Table 4 and the Appendix. Positive results were obtained even with sera that had been stored for long periods of time, which would be expected to reduce the IgE level (but not cause false readings). Follow-up of patients showed a gradual decline in IgE antibody levels, with RAST remaining positive for a minimum of 4 years and a maximum of > 17 years. Of the 34 patients with positive RAST with NMB, 11 had no history of previous anaesthesia and five had an uncertain anaesthetic history.

IgG RAST. This was performed in order to study the profile of the antibody response to NMB. It gave striking results that seemed to demonstrate the presence of antibodies to NMB (or QAG), particularly with ALC discs in everybody (allergic and controls, Fig. 3), including those who had never had a general anaesthetic.

RAST inhibition and elution. Choline, alcuronium and other compounds containing QAG only partly inhibited RAST (unlike HRL, with 100% inhibition) and eluted part of the serum antibody (IgE or IgG) that had been bound to allergen discs (after RAST).

Thiopentone RAST

IgE RAST. In normal patients mean and SD of uptake of labelled anti-IgE by THIO discs were 0.94 and 0.24% respectively (mean + 3 SD = 1.66%; higher figures considered positive). Nine patients had a positive thiopentone RAST, one with thiopentone only (no. 36, Appendix) and eight with both NMB and thiopentone. Only four of those nine patients showed histamine release (HRL) with thiopentone.

IgG RAST. This was positive both in allergic and normal subjects.

Skin tests

These were of limited diagnostic value, in general. Unless carried out with a range of concentrations, little information was obtained in the case of NMB, because false positive results were obtained frequently in the intradermal (i.d.) test in normal patients, particularly with tubocurarine and atracurium. False negatives occurred also in the prick test.³⁵ Furthermore, one of the allergic patients developed anaphylaxis after a 20-µg i.d. test dose of suxamethonium. A skin test with thiopentone was weakly positive in four patients (all with positive HRL and RAST) and negative in the rest. Skin tests with opioids were of no use because of false positives in normal patients.

Discussion

The results show that paper radioallergosorbent tests (RASTs) for IgE antibodies, particularly to neuromuscular blockers (NMB) are useful in the diagnosis of anaphylactic reactions associated with general anaesthesia. They also suggest that NMBs are the most common cause of such reactions. This trend is continuing. It seems that almost all

Table 4. Summary of results in patients who were investigated by the leucocyte histamine release (HRL), and radioallergosorbent (RAST, IgE) tests.

Period	1972-76	1977-81	1982-86	1987-89	Total
Number with positive HRL, mostly relaxants (all severe systemic reactions)	12*	8	8	8	36
<i>RAST positive with</i>					
Relaxants only	9	7	5	4	25
Thiopentone only	1	0	0	0	1
Relaxants + strong thiopentone	0	0	0	2	2
Relaxants + weak thiopentone	1	1†	2†	2†	6
Positive with relaxants only or mainly	10	8	7	8	33
Total positive (relaxant and (or) thiopentone)	11	8	7	8	34
Negative	1	0	1	0	2
Number with negative HRL (all less severe)	3	1	1	3	8
<i>RAST positive</i>					
Relaxants only	0	0	1	0	1
Thiopentone only or +relaxants	0	0	0	0	0

*These 12 did not include any patients with reactions to alphaxalone-alphadolone or propanidid. †Negative HRL with thiopentone.

cases with the so-called anaphylactoid reactions to NMB, including those with no history of exposure, are associated with significantly high levels of IgE antibodies to those agents, and thus are almost certainly truly anaphylactic. Paper-RAST with choline (CHOL) discs (commonly called 'suxamethonium' discs because of close chemical similarity, Fig. 1) appears to be the most simple and reliable test for IgE-antibodies and reactions to NMB. It was positive in 32 out of 35 patients showing histamine release from leucocytes (HRL). The agreement (positive or negative) between these two tests was noticed despite the fact that most of the samples were stored for long periods of time. Long storage

may explain the negative RASTs in two out of the 35 patients. All the samples of serum since 1987, whether from new or old patients at follow-up, were tested soon after collection, and this made no difference to the pattern of the results.

In a few patients there were gross quantitative discrepancies between IgE RAST and HRL (comparison to be published elsewhere), and between RAST and the severity of clinical reaction, e.g. two recent patients with severe clinical reaction had modest (but clearly abnormal) RAST results. High RAST was always associated with a severe reaction.

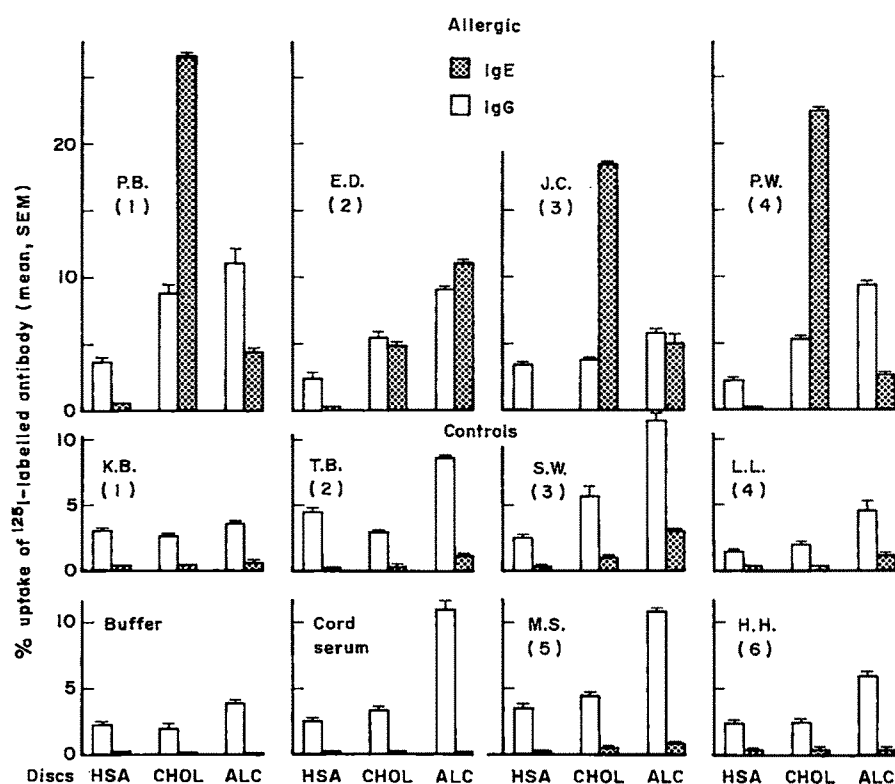


Fig. 3. Detection of IgE and IgG antibodies to the quaternary ammonium group and neuromuscular blockers by the paper radioallergosorbent test (RAST), using choline (CHOL) and alcuronium (ALC) allergen discs, and control discs (human serum albumin, HSA).

The apparently 'spontaneous' sensitisation to NMB in some patients and in experimental animals³⁸ may be because of prior exposure to crossreacting chemicals in the environment,^{32,39} or (more remotely) endogenous choline-containing substances ('autosensitisation'). The latter possibility and the apparent prevalence of 9:1 in females require further investigation.

Only four patients had proven allergy to thiopentone, in association with a stronger response to NMB in three, and on its own in one. The weakly positive thiopentone RASTs (five patients, all with positive NMB RAST) that were not associated with HRL or a positive skin test may be of little significance, may possibly be the result of a minor degree of crossreaction, and may have to be taken into consideration when setting the cut-off line between the normal and the genuinely allergic. They were not associated with grossly elevated total serum IgE, which has been suggested to cause false positives in a different thiopentone RAST.⁴⁰

The apparent detection of IgG antibodies in all sera with alcuronium (ALC) discs, and in most with choline (CHOL, Fig. 3) and thiopentone, whether allergic or control, and whether previously exposed to those drugs or not, is a most interesting finding, although difficult to explain. Allergen discs used in the IgE- and IgG-RAST are identical and it is unlikely that the detection of IgG antibodies is a disc-related artefact. These IgG antibodies have a number of precedents, e.g. in relation to penicillin.⁴¹

RAST studies on a 'pilot' scale are needed in deaths suspected of being associated with anaesthesia to seek evidence of anaesthetic allergy. Prospective studies are also needed to address the question of morbidity.

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Appendix

Summary of results of RAST in 36 patients with anaphylactic reaction during the induction of general anaesthesia. All received thiopentone and neuromuscular relaxants (NMB) and showed significant histamine release from leucocytes (HRL).

Patient, date of reaction and last follow-up	Previous exposure Y/N/? or	NMB given before reaction	Crossreaction other NMB	IgE RAST (% uptake* of ¹²⁵ I-anti-IgE by disc)		
				CHOL	ALC	THIO
Positive HRL, mainly NMB only (two also with THIO, numbers 7 and 10)						
1, 1981-82	Y	SUX	Y	26.4 (0.3)†	4.2 (0.3)†	1.0 (0)
2, 1985-89	N	ALC	N	4.8 (0.2)†	11.0 (0.2)†	2.1 (0.1)†
3, 1987-89	Y	SUX	Y	18.6 (0.3)†	4.9 (0.7)†	1.5 (0)
4, 1987 (fatal)	N	SUX	Y	22.3 (0.3)†	2.7 (0.2)	0.9 (0)
5, 1981-89	N	SUX	Y	12.2 (0.1)†	1.0 (0)	0.7 (0.1)
6, 1988	Y	SUX	Y	4.2 (0.1)†	1.6 (0)	1.5 (0.1)
7, 1988-89	Y	TUB	Y	5.0 (0.6)†	13.1 (0.9)†	9.5 (0.4)†
8, 1989	N	SUX	Y	8.1 (0.2)†	1.8 (0)	1.5 (0)
9, 1989	N	SUX	Y	5.1 (0)†	3.6 (0.2)†	1.8 (0)†
10, 1989	N	SUX	Y	4.2 (0.9)†	12.7 (0.3)†	9.7 (0.2)†
11, 1985-89	Y	SUX	Y	2.0 (0)†	4.7 (0)†	1.1 (0.1)
12, 1985-89	Y	ALC	N	2.9 (0)†	6.3 (0)†	3.5 (0.1)†
13, 1985-89	Y	SUX	Y	2.1 (0.2)†	1.7 (0.1)	1.4 (0.1)
14, 1985-89	Y	ALC	n.d.	2.9 (0.2)†	13.2 (0)†	2.3 (0.2)†
15, 1983	?	SUX, TUB	Y	3.8 (0)†	0.6 (0)	0.7 (0.1)
16, 1982-89	Y	ALC	N	0.4 (0)	7.5 (0.2)†	1.3 (0.1)
17, 1982	?	ALC	Y	4.3 (0)†	8.3 (0.1)†	1.2 (0.1)
18, 1982	N	SUX	Y	0.6 (0.1)	0.7 (0)	1.0 (0)
19, 1981-89	Y	SUX	Y	3.3 (0.2)†	3.0 (0)	2.1 (0.1)†
20, 1981-89	Y	SUX	Y	9.0 (0.1)†	2.2 (0)	0.7 (0.1)
21, 1979-81	N	SUX	Y	6.9 (0.1)†	13.6 (0.0)†	0.5 (0)
22, 1979-88	N	SUX	Y	6.1 (0.1)†	1.0 (0)	1.8 (0)†
23, 1977-89	Y	SUX	N	5.3 (0)†	3.2 (0.1)	0.8 (0.1)
24, 1977	?	SUX	N	4.1 (0.1)†	3.1 (0.1)	1.2 (0.1)
25, 1976	N	PAN	Y	4.4 (0.7)†	4.6 (0.5)†	1.7 (0)
26, 1976	Y	SUX, GAL	Y	3.5 (0.3)†	6.3 (0.3)†	1.3 (0.1)
27, 1976	N	SUX, PAN	Y	5.0 (0)†	0.2 (0)	1.2 (0.1)
28, 1976	N	ALC	Y	1.50 (0.2)†	1.0 (0.1)	1.2 (0)
29, 1976	?	PAN	Y	4.0 (0.1)†	1.6 (0.1)	1.3 (0)
30, 1975	?	SUX	Y	2.0 (0)†	4.7 (0)†	0.8 (0)
31, 1974	Y	PAN	Y	2.9 (0)†	0.6 (0)	1.2 (0.1)
32, 1973-89	Y	SUX	Y	1.8 (0)†	1.6 (0)	1.2 (0.1)
33, 1973-88	Y	SUX	Y	5.5 (0.3)†	1.8 (0.1)	0.8 (0)
34, 1973-89	Y	SUX	Y	8.7 (0)†	9.3 (0.3)†	2.7 (0.2)†
35, 1972-89	N	SUX	Y	0.7 (0)	1.1 (0)	0.9 (0.1)
Positive HRL with THIO only						
36, 1974	Y	SUX (+ THIO)		0.5 (0)	0.6 (0.1)	2.5 (0.1)†

SUX, suxamethonium; ALC, alcuronium; CHOL, choline; TUB, tubocurarine; PAN, pancuronium; GAL, gallamine. *% uptake of ¹²⁵I-labelled anti-IgE added to discs, mean (SEM) (done in triplicate to calculate measurement error). †positive (> grand mean + 3 SD for the group of normal subjects). n.d. = not done.

CASE REPORT

Aspiration pneumonia and the laryngeal mask airway

R. M. GRIFFIN AND I. S. HATCHER

Summary

A case of aspiration pneumonia is reported after the use of a laryngeal mask airway in a young woman undergoing an elective cholecystectomy. The case illustrates the hazards of regurgitation with the laryngeal mask airway and the need for further evaluation when used with controlled mechanical ventilation.

Key words

*Equipment; laryngeal mask airway.
Complications; aspiration.*

The laryngeal mask airway was introduced as an alternative to conventional mask anaesthesia or tracheal intubation. Initial experience with the laryngeal mask airway suggested that it could be used successfully for controlled mechanical ventilation of the lungs.^{1,2} Concern still exists about its use for patients in whom there is an increased risk of inhalation of gastric contents.³ Currently the manufacturer's data sheet reflects this opinion and states specifically that 'it does not prevent regurgitation'.

There are no reports in the anaesthetic literature, to our knowledge, of significant aspiration pneumonia after the use of laryngeal mask airway. Recent correspondence has provided anecdotal evidence of regurgitation,⁴ but in these instances a significant aspiration pneumonia did not occur. We report a case in a previously healthy young woman undergoing elective surgery.

Case history

A 60-kg, 26-year-old woman with no significant previous medical history was scheduled for an elective cholecystectomy after an attack of cholecystitis 3 weeks before. She was premedicated with temazepam 20 mg one hour before operation. Anaesthesia was induced with fentanyl 100 µg, droperidol 2.5 mg, thiopentone 250 mg and vecuronium 6 mg. A size 3 laryngeal mask airway was passed easily after adequate relaxation, and the cuff inflated with 20 ml of air to obtain a gas-tight seal.

Anaesthesia was maintained with enflurane (1%) in a mixture of 66% nitrous oxide and 33% oxygen. A Siemens 710 ventilator was set to give a tidal volume of 430 ml and an inflation pressure of no more than 1.5 kPa; expired

minute volume equated with inspired minute volume that was set at 6 litres. Oxygen saturation and the electrocardiogram (lead II) were monitored continuously and noninvasive blood pressure intermittently (Hewlett Packard 78352A). The degree of neuromuscular block was monitored with a Bard nerve stimulator and increments of vecuronium (1 mg) given when the block decreased below 75%. Total duration of surgery was 1.5 hours.

The patient was placed in a left lateral position, on completion of surgery, in order to perform right intercostal nerve blocks (T₄₋₈) with 0.5% bupivacaine. Spontaneous ventilation returned without the need for neuromuscular reversal, as confirmed by nerve stimulation.

The cuff was deflated before removal of the laryngeal mask airway. Immediately, bile-stained fluid was noted within the lumen. The airway was removed at this point and a small volume of similar fluid was found within the aperture. The patient was immediately placed in a head-down position and the pharynx sucked out. Oxygen saturation decreased to 87% on air, but this rapidly improved with administration of oxygen via a Hudson mask. The patient was awake at this point and complaining of shortness of breath with retrosternal discomfort.

She was transferred to the Intensive Care Unit for further monitoring and treatment. Chest X ray performed on admission confirmed the diagnosis of a significant left-sided aspiration pneumonia. There was very extensive patchy consolidation present throughout the left lung with slight elevation of the left hemidiaphragm.

The patient was treated with oxygen, initially with an inspired concentration of 60%, and antibiotic cover continued with cefuroxime and metronidazole. Symptoms of

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Accepted 4 April 1990.

respiratory distress had resolved by the time of admission to the ICU and arterial blood gases were pH 7.43 P_{CO_2} 4.2 kPa, P_{O_2} 35.4. A thoracic epidural was sited and a fentanyl infusion commenced for additional analgesia to facilitate effective chest physiotherapy, since the patient was still experiencing some discomfort.

The inspired oxygen concentration over the next 24 hours was progressively reduced and the patient complained of no discomfort or difficulty in breathing. The arterial blood gases on air on discharge from the ICU were pH 7.42, P_{CO_2} 4.6 kPa, P_{O_2} 11.5 kPa. Intermittent blood gas analysis remained satisfactory on the ward and the patient was discharged after 9 days with partial resolution of the radiographic appearances.

Discussion

Aspiration pneumonia remains a potentially serious problem during anaesthesia,⁵ although there are surprisingly few published data on the incidence and predisposing risk factors. One large study reported an incidence of 1 in 2131 anaesthetics,⁶ with children and the elderly most often affected. In the majority of cases one or more pre-operative risk factors were identified, which may have predisposed to aspiration. These included emergency operation, upper abdominal surgery, a history indicative of delayed gastric emptying, unusual stress or pain. There were no features in our patient, other than that she was having an upper abdominal procedure, to indicate that she was at increased risk of regurgitation.

It is the recent routine practice of one of the authors (R.G.) to use a laryngeal mask airway, without complication, to provide ventilation of the lungs during upper abdominal surgery. Regurgitation on this occasion resulted presumably from gastric distension caused by a gas leak around the airway that was not of such a proportion to be detected by a discrepancy between the inspired and expired minute volumes. In combination with the upper abdominal surgery the lower oesophageal sphincter became incompetent and allowed reflux of gastric contents to occur.

Reservation was expressed about use of the laryngeal mask airway for ventilation of the lungs.³ A nasogastric tube may allow the escape of any gases blown into the stomach during ventilation but may produce malocclusion of the laryngeal mask cuff to the pharyngeal walls, thereby increasing any anaesthetic gas leak. Furthermore, a nasogastric tube renders the lower oesophageal sphincter

incompetent, and allows gastric contents to regurgitate around the nasogastric tube more easily. These factors would appear to militate against the use of a nasogastric tube with the airway.

The manufacturer's data sheet does make a cautionary note that the laryngeal mask airway does not prevent regurgitation.⁷ Payne,⁸ using fiberoptic laryngoscopy, noted in three patients from a study cohort of 50, that the oesophagus was clearly visible with the airway *in situ*. In the present case, a collection of regurgitated fluid was found within the aperture after removal. The deflated cuff may have obstructed the egress of gastric contents out of the oesophagus and, instead, directed these into the larynx and down the trachea. Other authors have made a similar observation,^{9,10} which confirms our findings, and suggests that the laryngeal mask airway actually may predispose to aspiration of any regurgitated material.

The timing of cuff deflation and airway removal, a manoeuvre often performed by recovery staff, appears to be critical if regurgitation and aspiration are to be avoided. It is evident that for regurgitation and aspiration to have occurred the patient was not awake enough to protect her own airway. However, she was alert and cooperative very shortly after the airway was removed. We emphasise the importance of leaving the cuff fully inflated until the patient is swallowing and almost awake.

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Severe anaphylactoid reaction to hydroxyethyl starch

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Summary

A case is presented of a severe anaphylactoid reaction to hydroxyethyl starch solution that occurred peri-operatively and required extended intensive management of the resultant bronchospasm and hypotension. Subsequent intradermal injection of hetastarch produced a delayed positive response, suggestive of a complement-mediated mechanism.

Key words

Blood; replacement, hydroxyethyl starch.

Allergy; anaphylaxis.

Hydroxyethyl starch (or hetastarch), a synthetic colloid derived from amylopectin, is used increasingly as a plasma substitute. Hetastarch is in clinical use as a 6% solution (Hespan, Du Pont, Stevenage, UK). It has an average molecular weight of 70 000 with 95% of molecules falling within a range of 10 000–1 000 000 dalton, and a prolonged intravascular life in comparison with other synthetic colloids.¹ Nonantigenicity² and lack of association with histamine release³ are among the main advantages of hetastarch. Ring and Messmer investigated prospectively the frequency of anaphylactoid reactions to colloids in 1977 and found a 0.085% incidence of reactions to hetastarch; the incidence of severe reactions was 0.006%.⁴ It is not known whether these are due to the hydroxyethyl starch itself or to the trace impurities present in the hetastarch solution.

We report the case of a severe anaphylactoid reaction, corresponding to Grade III on the Ring and Messmer severity scale³ (i.e. shock, life-threatening spasm of smooth muscle, etc.), to hetastarch that required prolonged treatment with adrenaline.

Case history

A 76-year-old woman presented with haemoptysis. She had a history of recurrent haemoptysis secondary to bronchiectasis and long-standing atrial fibrillation controlled by digoxin, but was otherwise fit and well and receiving no other medication. She had no history of allergy and there was no family history of note. She suffered a further

haemoptysis in the local Casualty Department followed by a respiratory and then cardiac arrest. She was resuscitated rapidly, with 500 ml polygeline (Haemaccel, Hoechst, Brentford, UK) without incident. She was transferred to the Middlesex Hospital the next day for left upper lobectomy.

Her lungs were ventilated mechanically and she was sedated overnight in the intensive therapy unit (ITU) where she received an infusion of papaveretum and occasional boluses of midazolam. She was haemodynamically stable with a blood pressure of 120/60 mmHg and passed adequate quantities of urine. Anaesthesia for lobectomy was induced with propofol 100 mg, fentanyl 100 µg and vecuronium 6 mg. Compound sodium lactate solution was infused slowly through a peripheral venous cannula. Anaesthesia was maintained with 50% oxygen, 50% nitrous oxide and 1% enflurane by intermittent positive pressure ventilation. An uneventful left upper lobectomy was performed; the procedure lasted approximately 90 minutes. The patient received further increments of vecuronium 2 mg and fentanyl 50 µg after 40 minutes, at which time the papaveretum infusion was restarted. No further drugs were given until the end of the procedure when 0.5% bupivacaine was used for intercostal nerve blocks.

Systemic arterial pressure decreased towards the end of the operation from 120/60 to 90/60 mmHg and central venous pressure fell slightly from 7 to 6 mmHg over 5 minutes. Estimated blood loss was 400 ml, and an infusion of hetastarch was started through the peripheral cannula. The arterial pressure continued to decline slowly and the area around the infusion site appeared oedematous. The

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Accepted 1 May 1990.

peripheral infusion was assumed to have leaked into the tissues, so the hetastarch was infused briskly through a central venous line. There was an immediate decrease in systolic arterial pressure to 40 mmHg and peak inflation pressure increased from 2 to 4 kPa with audible wheeze over both lung fields. The infusion arm was noted to be both erythematous and oedematous 5 minutes later, and over the next 5 minutes this had spread to the patient's face. The hetastarch infusion was stopped; however, 150 ml had already been given through the peripheral line and almost all of the remaining 350 ml through the central line. An intravenous bolus injection of adrenaline 0.4 mg restored the arterial pressure for only a few minutes. A second dose was administered and an infusion given at a rate of 3–5 µg/minute. Chlorpheniramine 10 mg, ranitidine 50 mg, hydrocortisone 200 mg and aminophylline 100 mg were also given intravenously. The patient was returned to the ITU where mechanical ventilation and the adrenaline infusion were continued.

The rash and oedema resolved within one hour but the severe bronchospasm continued and her arterial pressure remained adrenaline-dependent. She was managed with fentanyl sedation, salbutamol nebulisers, hydrocortisone, adrenaline and histamine antagonists. The adrenaline infusion could be discontinued only 24 hours after the initial incident. By this time the peak inflation pressures had also returned gradually to pre-operative values. Her trachea was extubated later that day and she made an uneventful recovery.

On local advice, blood samples were not withdrawn at the time of the incident. A late sample (taken approximately 3 weeks later) was analysed after subsequent discussion with the National Adverse Anaesthetic Reactions Advisory Service. Complement levels were slightly raised. Plasma IgE levels were increased to an atopic level of 1000 units/ml (approximately 100 times normal). IgG, IgA and IgM levels were also above normal but these may have been related to her long-standing bronchiectasis.

Hetastarch was presumed to have been responsible for the anaphylactoid reaction because of the close temporal relationship between administration of this product and the onset of signs and symptoms. The patient returned for skin-prick and intradermal testing 2 months later. The bag of hetastarch used peri-operatively was mislaid, but one bag of solution, and also samples of hetastarch powder used to constitute some of the different batches of the 6% solution in use in the hospital, were provided by the manufacturers. Skin prick testing on the forearm was negative. Intradermal injection of a 0.05 ml saline control produced no reaction. This was repeated using 0.05 ml of a 1:10 dilution of the hetastarch solution followed, 10 minutes later, by 0.05 ml of normal-strength hetastarch solution. No initial reaction was seen to the normal hetastarch solution, but after 10 minutes a typical wheal and flare reaction measuring 10 mm in diameter developed. At this time a milder reaction of 2-mm diameter was noted over the injection site of the 1:10 diluted hetastarch.

Discussion

All colloid solutions can cause varying degrees of allergic reaction. Hydroxyethyl starch, as mentioned previously, is associated with a low incidence of severe (Grade III or Grade IV) reaction. Ring and Messmer⁴ recorded only one Grade III reaction to hetastarch but did not provide specific details. Porter and Goldberg⁵ described a patient who developed hypotension, urticaria and angioedema, but no bronchospasm, on receiving hetastarch soon after induction of anaesthesia. No adrenaline was needed and the patient's trachea was extubated after 10 hours without difficulty. They found slightly depressed concentrations of complement CH50 but normal concentrations of histamine, and suggested a complement-mediated mechanism. Our finding of a delayed intradermal reaction adds further weight to this theory, especially as there was no evidence of previous exposure to hetastarch.

The patient required prolonged treatment with adrenaline to control both the hypotension and the bronchospasm; this is in keeping with the prolonged intravascular life of hetastarch. Possible explanations for this type of anaphylactoid reaction include the large molecular weight fractions of hetastarch or trace impurities. Unfortunately, the patient declined to attend for intradermal testing of further hetastarch solutions of varying molecular size and manufacture source.

This is the first recorded case of an extended anaphylactoid reaction to hetastarch solution, and although such a reaction is extremely rare, clinicians should be aware of its existence and the possible need for prolonged treatment.

Acknowledgments

We thank J. Fuller and R. Carter of Du Pont (UK) Ltd and Dr J. Watkins, Coordinator of the National Adverse Anaesthetic Reactions Advisory Service, Department of Immunology, Royal Hallamshire Hospital, Sheffield for their invaluable assistance. We also thank Mr T. Treasure and Dr N. Johnson for their cooperation.

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CASE REPORT

Propofol infusion for control of status epilepticus

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Summary

Two patients with status epilepticus who were resistant to conventional treatment but responded to propofol infusions are reported. An electroencephalogram confirmed the seizures and their successful treatment.

Key words

*Anaesthetics, intravenous; propofol.
Complications; status epilepticus.*

Considerable controversy surrounds the effect of propofol on seizure activity. There were reports, unsubstantiated by electroencephalography (EEG) of epileptic seizures after propofol administration.¹ Conversely its use has been described in the treatment of status epilepticus,^{2–4} but in none of these was there confirmation of anticonvulsant action by full EEG recordings both before and after treatment. We describe two cases of unusual forms of status epilepticus, which had not responded to conventional anticonvulsant therapy, but in which propofol successfully controlled the seizures. Successful treatment is confirmed by EEG recordings before and after treatment.

Case histories

Case 1

A previously healthy 19-year-old woman presented with a subacute febrile illness associated with extreme agitation, dystonic movements, focal twitches and apnoeic spells. A diagnosis of encephalitis lethargica was made. She was treated empirically with acyclovir, penicillin and phenytoin. Her EEG in the course of her illness showed a left-sided discharging focus, coinciding with right lower limb clonic twitching. Her seizures persisted, despite the addition of diazepam, clonazepam and phenobarbitone to her drug regimen. A thiopentone infusion was started and mechanical ventilation instituted. Her EEG was monitored during thiopentone administration until a burst suppression pattern was achieved. This was maintained for 3 days, at which time she became hypotensive and thiopentone was withdrawn. Thereafter she remained unresponsive for 4

days. Her EEGs, after thiopentone withdrawal, showed a recurrence of asymmetry, with intermittent sharp and slow wave complexes noted, especially on the right side (Fig. 1). Initially the EEG changes were merely observed, but when clinical seizures recurred, propofol was started at 6 (mg/kg)/hour and increased to between 7 and 10 (mg/kg)/hour until her seizures were controlled. This was confirmed on an EEG performed 12 hours after the start of the infusion, which showed well formed alpha activity with no evidence of seizures (Fig. 2). The propofol dosage was gradually reduced after a few days and withdrawn completely after 12 days. Further EEGs showed a return of beta activity but with no return of seizures. She went on to make a slow but almost complete recovery. Other aspects of this case were reported elsewhere.⁵

Case 2

A 26-year-old man with chronic renal failure and tertiary hyperparathyroidism developed severe and refractory hypocalcaemia after parathyroidectomy. His serum calcium persisted between 1.55 and 1.71 mmol/litre despite large doses of calcium and vitamin D. His renal function deteriorated with a rising blood urea and creatinine, hyperkalaemia and hypertension (250/150 mmHg). He had a grand mal fit on the 15th postoperative day, which was treated with diazepam and phenytoin. He was also given calcium gluconate because of his persistent hypocalcaemia, and labetalol for hypertension. He was transferred to the Intensive Therapy Unit with a provisional diagnosis of hypertensive encephalopathy. He remained unresponsive after treatment but with generalised hyper-reflexia. An

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Accepted 21 March 1990.

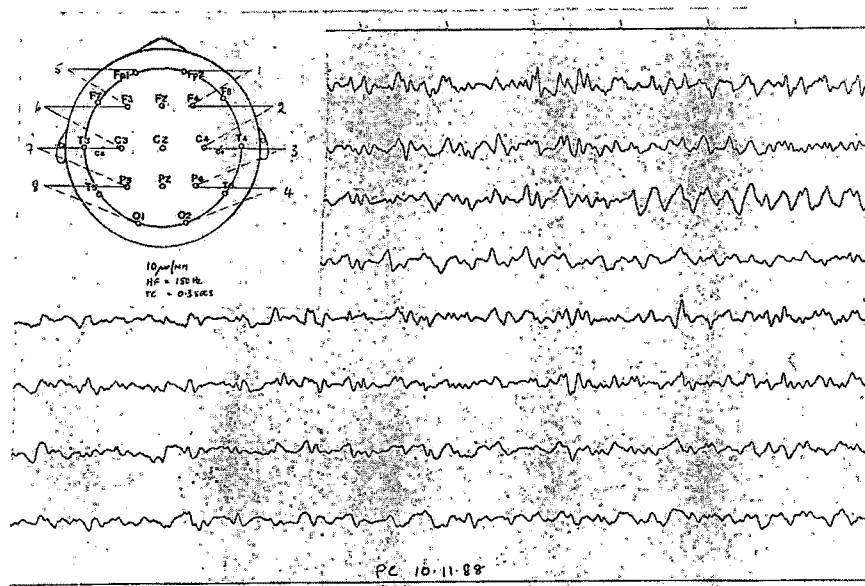


Fig. 1. Case 1. EEG before propofol administration. Intermittent sharp and slow wave complexes, predominantly right sided.

EEG showed bifrontal sharp waves that coincided with almost imperceptible movements of his fingers and toes (Fig. 3). A diagnosis of nonconvulsive status epilepticus secondary to either hypocalcaemia or hypertension was made. The seizure activity did not respond to further diazepam or phenytoin. A CT scan was normal. His conscious level deteriorated and his respiration became inadequate; mechanical ventilation was commenced. Under EEG monitoring intravenous propofol 2 mg/kg was shown to abolish his seizure activity and he was therefore continued on an infusion of propofol at 7 mg/kg/hour. This maintained a burst suppression pattern on his EEG (Fig.

4). He continued to receive calcium, vitamin D and labetalol and required haemofiltration for his worsening renal failure. Propofol was withdrawn after 5 days and his conscious level gradually improved with no return of seizure activity. His renal function also improved and he made a complete recovery.

Discussion

Propofol is a useful drug for sedation of patients in the Intensive Therapy Unit,⁶ but its role in seizure disorders is controversial. Convulsions and involuntary movements

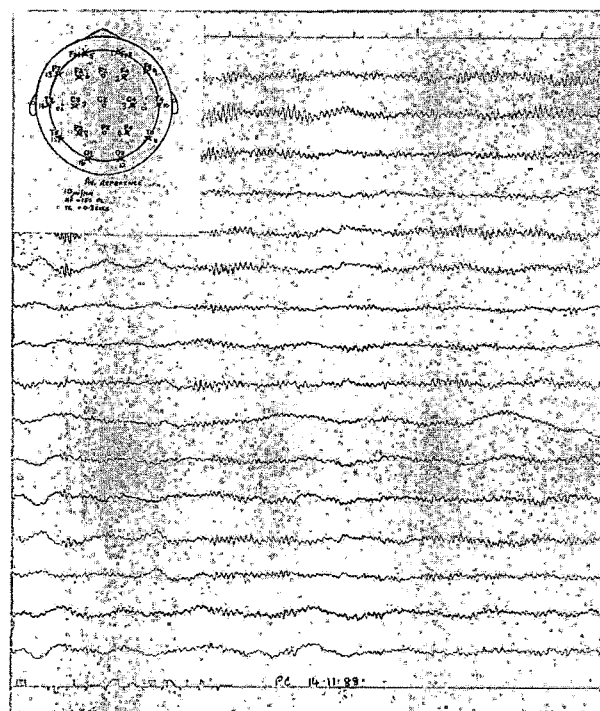


Fig. 2. Case 1. EEG during infusion of propofol at 8 mg/kg/hour. Alpha rhythm is seen with no evidence of seizure activity.

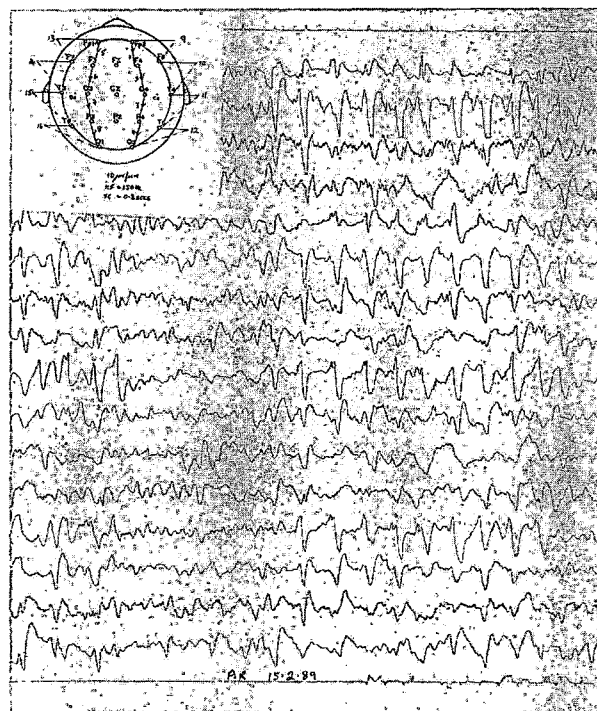


Fig. 3. Case 2. EEG before propofol administration. Bifrontal sharp wave activity.

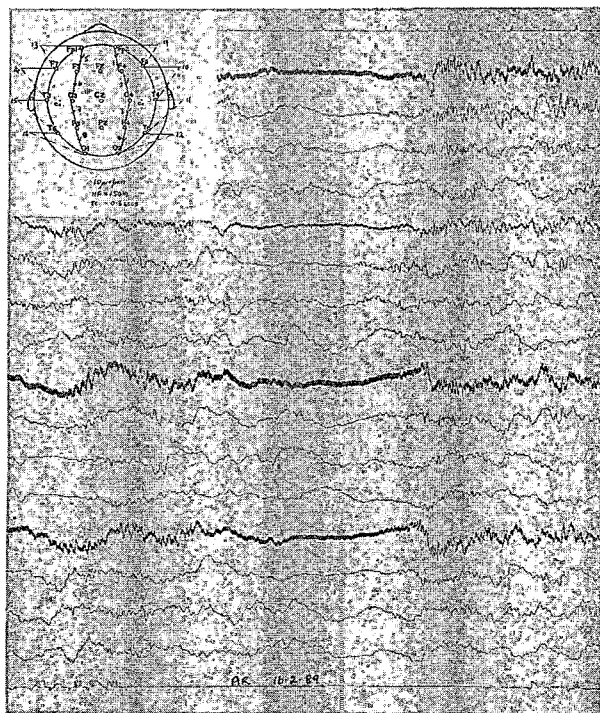


Fig. 4. Case 2. EEG during infusion of propofol at 7 mg/kg/hour demonstrating burst suppression.

were described during induction of, and emergence from, anaesthesia, in both epileptic and nonepileptic patients.¹ On the other hand there have been reports of propofol successfully controlling status epilepticus unresponsive to conventional therapy.²⁻⁴ The suggestion that the duration of seizures is shortened when propofol is used for anaesthesia for electroconvulsive therapy also supports an anticonvulsant effect⁷ as does some animal work, where fits were induced by injection of pentylenetetrazol or by electroshock.⁸

It remains difficult to reconcile all the reported effects of propofol on seizure activity. One explanation is that the clinical result depends on the balance between effects on

inhibitory and excitatory neurones, as occurs with local anaesthetics, and that this might be dose related. It is important that reports should include EEG confirmation to avoid any possibility for clinical misdiagnosis.

The two cases reported here provide objective evidence of an anticonvulsant effect, with seizure activity demonstrated on EEG before the abolition of propofol administration. Both cases represent unusual forms of epilepsy, resistant to conventional therapy. Thiopentone was effective in the first case as an anticonvulsant, but hypotension and oliguria necessitated its withdrawal, which was followed by a delayed recovery of consciousness. These problems were not seen with propofol.

We believe that propofol is an effective anticonvulsant that should be considered when status epilepticus does not respond to conventional first-line therapy. Its use will, of course, require mechanical ventilation and intensive cardiovascular and EEG monitoring.

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Pregnancy associated with Gorlin's syndrome

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Summary

A pregnant woman with Gorlin's syndrome presented for the removal of malignant ovarian tumours. The main problem encountered by the anaesthetists was an unexpected increase in arterial blood pressure. This was most probably associated with surgical manipulation of the ovaries with an increase in prorenin/renin production.

Key words

*Complications; Gorlin's syndrome.
Pregnancy.*

Gorlin and Goltz¹ described in 1960 a syndrome of multiple basal cell naevi associated with other anatomical abnormalities which have included jaw cysts, bifid ribs and occasionally pectus excavatum, meningiomata, oculoneurological abnormalities and ovarian tumours. The syndrome is inherited as an autosomal dominant with variable expressivity and high penetrance and appears to follow a similar course within single families.

Three major complications are recorded associated with the induction of anaesthesia in these patients: acute hypotension, bradycardia and bronchospasm.² It is tempting to believe that they are the result of an allergic type of reaction, since some of these complications occurred at a subsequent general anaesthetic with one of the intravenous anaesthetics acting as the 'triggering agent'. We encountered a different complication, previously unreported, which affected the conduct of anaesthesia and was most probably related to ovarian prorenin/renin production.

Case history

A 31-year-old female was admitted with a 3-year history of primary infertility but was now 17 weeks' pregnant. She had had an uneventful laparoscopy under general anaesthesia 2 years previously when her ovaries were found to be enlarged with small cysts but both Fallopian tubes were patent. General anaesthesia at that time included thiopentone, atracurium and fentanyl.

Palpation at a routine antenatal visit revealed large bilateral lower abdominal masses. Ultrasound echography suggested that these were malignant tumours which originated from both ovaries. She had also noticed her face

becoming hirsute and her voice deepening after the 13th week of gestation. Plasma testosterone level was 92.4 nmol/litre (normal range 0.3–2.5 nmol/litre) at the 15th week of gestation. Further ultrasound echography at the 17th week showed one normal sized fetus, a 9-cm diameter mass on the right side and an 8-cm diameter mass on the left.

She had small scarred areas on her face, chest and back on examination which were the result of treatment with liquid nitrogen to malignant basal naevi over the last 6 months. A jaw cyst and T₃ hemivertebra with bifid ribs were found on the X ray but there were no clinical symptoms. The findings were consistent with Gorlin's syndrome, although none of her family was affected. Her height and weight were 171 cm and 73 kg respectively. Arterial blood pressure was 160/110 mmHg with a regular pulse rate of 100 beats/minute. Her upper respiratory tract looked normal (Mallampati class I).³ The abdominal size corresponded to a 32–34 week pregnancy. The haemoglobin was 12.9 g/dl, and the urea and electrolyte values were within the normal range.

Anaesthetic management

Lorazepam 2 mg orally was given 1.5 hours before operation. The ECG, indirect arterial blood pressure and oxygen saturation were monitored continuously in the anaesthetic room. A pillow was put under the left buttock, an intravenous line inserted and 500 ml of compound sodium lactate solution given before induction. The heart rate and arterial blood pressure were 120 beats/minute and 160/100 mmHg respectively. Anaesthesia, after pre-oxygenation, was induced with fentanyl 100 µg, etomidate 16 mg and

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Accepted 23 February 1990.

suxamethonium 50 mg and tracheal intubation was easily achieved. Anaesthesia was maintained with 1–2% halothane in 33% oxygen in nitrous oxide with supplementary doses of fentanyl and pancuronium.

The operation continued uneventfully for 15 minutes. However, when the surgeons handled the ovarian tumours, the arterial pressure, which had decreased to 130/90 mmHg, rapidly increased to 160/110 mmHg and the heart rate to 130 beats/minute despite an increase in the halothane to 2% and another 100 µg of fentanyl. The arterial pressure and heart rate returned to previous levels after the ovarian tumours were removed. The operation lasted 1.5 hours, during which 1000 ml of blood were lost and she was given 1000 ml of compound sodium lactate solution, 500 ml of hydroxyethyl starch and two units of concentrated red cells. Her trachea was extubated uneventfully after the reversal of residual neuromuscular blockade.

Postoperative course

The postoperative period was uneventful but she received continuous intravenous ritodrine for the first 4 days to prevent uterine contractions. This treatment also produced hypertension and tachycardia. The blood pressure and heart rate returned quickly to average values when this therapy was stopped. She was discharged from the hospital, still pregnant, 10 days after the operation and a baby boy was delivered prematurely at 27 weeks' gestation. The baby developed respiratory distress syndrome which was successfully treated. Unfortunately, it is confirmed that he too has Gorlin's syndrome.

Hormonal study

Her plasma testosterone, oestradiol and progesterone levels were measured before operation, just before the tumours were removed, 6 hours, 24 hours and 4 days thereafter. All the levels were very high before and during surgery, although they increased very rapidly when the ovarian tumours were handled, rising by approximately 50% above pre-operative values. They returned to normal very quickly after surgery.

Conventional histopathology did not explain the events that occurred under general anaesthesia. However, immunohistochemical staining revealed the presence of prorenin/renin in the tumour cell and this was confirmed using two different polyclonal antirenin antibodies. A rabbit antibody⁴ raised against human renin completely purified from a juxtaglomerular cell tumour was applied to formalin-fixed paraffin-embedded tissue sections (5 µ thick) in conjunction with an indirect immunoperoxidase technique, the DNP-hapten sandwich staining procedure (DHSS).⁵ The resulting brown immunostaining was seen as finely granular deposits restricted to the cytoplasm of small tumour cells scattered around capillary/lymphatic vessels. This particular staining reaction was quite independently reproduced and utilised two antibodies, one of which was applied in conjunction with the peroxidase-antiperoxidase method.

Discussion

The first concern in the management of this patient was to try and avoid the complications that were reported during

previous anaesthetics. The second concern was a possible difficult tracheal intubation associated with the minor anatomical abnormalities of the airway.

The uterus was commensurate with a 17-week pregnancy, although the two additional large masses had greatly distorted the abdomen. It was therefore thought prudent to treat her like a full term pregnant patient and hence a support was placed under her left buttock and she received 0.5 litres of compound sodium lactate solution intravenously before induction.

The induction agents were chosen both for their cardiovascular stability and lack of histamine release. A small test dose of etomidate produced no abnormal reaction and was therefore used and she was managed like a term pregnant patient. Halothane was used because it inhibits uterine contraction, which is a positive advantage since surgery was to be undertaken in the vicinity of the uterus. Further, since it also causes bronchodilatation, it seemed the agent of choice. The fluid replacement of compound sodium lactate solution, hydroxyethyl starch and two units of concentrated red cells were also chosen to minimise the chances of a 'histamine release' reaction.

The rapid increase in all three hormones during surgical manipulation of the ovarian tumours coincided with both the increase in heart rate and arterial blood pressure. It seems likely that the one was related to the other either directly or indirectly. It is also likely that either prorenin or renin was released from the tumours during surgical manipulation. This would have increased the conversion of angiotensinogen to angiotensin I, hence producing increased amounts of angiotensin II; the latter would have increased the arterial pressure. Suitable prophylactic measures can be used to prevent this complication if the anaesthetist anticipates that this might happen. Recently angiotensin converting enzyme (ACE) inhibitors have been successfully used both orally⁶ and intravenously⁷ to prevent the hypertensive response to tracheal intubation. Such therapy would be useful if prorenin or renin is released into the blood stream.

The anaesthetic technique was chosen to reduce the possibility of the anticipated complications previously reported during induction of anaesthesia, although the rapid increase in blood pressure after the manipulation of the masses during surgery was not anticipated. This aspect may be of greater importance and needs to be borne in mind with such cases.

Acknowledgments

The authors thank Mrs S. Walker, FRCOG, for permission to present this case and both Professor M. Scanlon, FRCP, and Professor M.D. Vickers, FCAnaes for their helpful advice and encouragement. We are also very grateful to Dr G.B.M. Lindop (Glasgow) for his expertise.

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CASE REPORT

Atlanto-axial subluxation in Down's syndrome

J. F. POWELL, T. WOODCOCK AND F. E. LUSCOMBE

Summary

A case of atlanto-axial subluxation in a patient with Down's syndrome is described. The gradual deterioration in the patient's locomotor ability caused a delay in diagnosis. Patterns of presentation of this condition are discussed.

Key words

Anatomy; vertebral column.

Complications; Down's syndrome.

Physical anomalies associated with Down's syndrome can give rise to special problems for the anaesthetist. The danger of congenital cervical spine abnormality was recently discussed in relation to participation in strenuous sport,¹ but its relevance to anaesthesia is still not widely appreciated. We report a case of atlanto-axial instability associated with Down's syndrome in which a chronic deterioration in the patient's physical condition was wrongly attributed to severe mental handicap, which confused and delayed the final diagnosis.

Case history

An 18-year-old female with Down's syndrome was referred to the Intensive Care Unit for the management of acute respiratory failure attributed to postoperative pneumococcal pneumonia. The relevant past medical history obtained from the referring anaesthetist and the patient's guardian at this time was that the patient was severely physically and mentally handicapped and was confined to a wheelchair. She was admitted to the referring hospital for bilateral tendo Achilles elongation. On admission, the lower limb reflexes were noted to be abnormally brisk. The surgical procedure was performed under general anaesthesia with the patient breathing spontaneously after intubation with a cuffed tracheal tube. The procedure lasted approximately 15 minutes and the peri-operative course was uneventful.

She suddenly became cyanosed, tachypnoeic and hypotonic 40 hours after operation. The trachea was intubated

and the lungs ventilated, and rigid bronchoscopy performed. Mucopurulent sputum was aspirated from the right main bronchus and was sent to the laboratory for microscopy, culture and sensitivity. She was given intravenous cefuroxime and transfer to the ITU was requested. Examination on arrival showed a 25-kg female with Down's syndrome, whose conscious level was depressed by sedation with midazolam. She grimaced when moved, had some flexion of her right arm in response to painful stimulus, but showed no movement of her left arm or legs. The cardiovascular system was stable without inotropic support, but she had a pansystolic murmur at the left sternal edge and subsequent echocardiography showed a single perimembraneous ventricular septal defect with a left to right shunt. Chest X ray revealed bilateral midzone shadowing, and initial arterial blood gas analysis showed her to be hypoxaemic, P_{aO_2} 6.4 kPa, F_{iO_2} 0.4.

Her guardian revealed that she had the language development of a 2-year-old and could communicate in simple terms using Makaton. She had been able to walk independently and feed herself 2 years before her admission for operation, but her mobility at that time was deteriorating and she was referred for an orthopaedic opinion. Findings on examination then included spasticity in all limbs, flexion contracture of the right hip and knees, instability of the right knee and a tight right tendo Achilles. Radiographs of the right knee showed marked subluxation secondary to soft tissue laxity. An arthrodesis of the knee was planned when full growth had been attained; in the meantime elongation of tendo Achilles was recommended.

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Accepted 4 April 1990.

Her mobility continued to deteriorate and a year later she was wheelchair-bound. She was admitted then for evaluation, and marked hypotonia and hyperreflexia were again noted and attributed to her severe mental handicap. She received physiotherapy to improve her manual dexterity, but very little improvement was seen in her mobility. The Intensive Care Team did not suspect an acute cord lesion on the basis of the above history of a gradually progressive physical deterioration, and with a documented purulent pneumonia to account for her acute respiratory failure, and so they applied a soft cervical collar while the patient was sedated in case of atlanto-axial instability.

Sputum culture confirmed pneumococcal infection sensitive to cefuroxime, so this antibiotic and IPPV were continued. Sedation had been withdrawn by day two of Intensive Care and ventilation was satisfactory with synchronised intermittent mandatory ventilation (SIMV) plus pressure-supported spontaneous breaths. The mandatory rate was reduced to five breaths per minute by day five. The pneumonia had clinically and radiologically resolved, and she was disconnected from the ventilator. She was, however, tachypnoeic with inadequate tidal volumes, nasal flaring and intercostal recession. SIMV with pressure support was restarted. Her respiratory pattern off the ventilator was no better on day six and diaphragmatic paralysis was suspected. There was no evidence for a new cervical cord injury in the opinion of the medical team who reviewed her on day six and SIMV and pressure support were continued. Somatosensory evoked potentials were performed on day 10 and showed intact dorsal column conduction. She was still tachypnoeic and distressed, however, when disconnected from the ventilator.

Neurological opinion was sought on day 12; the patient was escorted to the X ray department and radiographs of her cervical spine obtained (Fig. 1). 'O' is the odontoid process of the axis, 'A' the anterior arch of the atlas and 'P' the posterior arch. These demonstrated craniovertebral

settling with atlanto-axial subluxation, and the sagittal canal diameter in extension was only 2.5 mm. The diagnosis of anterior spinal cord compression was confirmed, and it was believed that the neurological damage was by then irreversible and, after careful consideration, it was decided that attempts at neurosurgical decompression with spinal fusion were not indicated in this case.

Discussion

John Langdon Down first published the review of the syndrome that now bears his name in 1866. The two most common signs he described were mental retardation and hypotonia, but in the 1960s the increased incidence of atlanto-axial instability in Down's syndrome was reported. Tishler and Martel examined radiographs of the cervical spines of 18 individuals with the syndrome after their discovery of an atlanto-odontoid interval of 8 mm in a 10-year-old asymptomatic girl with Down's syndrome.² In four cases the atlanto-odontoid interval, measured at the inferior aspect of the anterior atlanto-odontoid joint, was greater than 5 mm. Pueschel found that if the atlanto-odontoid interval was 4.5–6.0 mm the patients remained free of neurological signs, but if the distance exceeded 7.0 mm all patients had neurological signs.³ The average distance between the anterior portion of the atlas and the anterior surface of the odontoid process of the axis, when examined radiographically, is up to 4 mm in normal children and less in adults. Figure 2 (a) and (b) illustrate atlanto-axial instability in flexion, with an extension view for comparison in another, adult, patient. The incidence of asymptomatic atlanto-axial instability in the general population is not known, but in a recent review, Collacott estimated the prevalence of atlanto-axial instability as between 12 and 22% in individuals with Down's syndrome.⁴ The incidence of neurological problems associated with, and probably secondary to, atlanto-axial instability is thought to be 2–3% of all patients with Down's syndrome who survive infancy.⁴ Symptomatic atlanto-axial instability in Down's syndrome was reviewed by Davidson⁵ after the recommendation, in a Special Olympics Bulletin in 1983, that competitors with Down's syndrome should be restricted from participating in events that might result in hyperextension of the neck. He found reports of 31 individuals and tabulated their presentations, and concluded that not only was the complication uncommon but that 'there is no evidence that the current roentgenographic criteria of atlanto-axial instability are predictive of a tendency to dislocation'. Pueschel disputes this view and quotes his own study of 404 individuals where 1.5% had symptomatic atlanto-axial instability.⁶ The cause for the increased incidence of atlanto-axial instability is thought to be due, in part, to the generalised ligamentous laxity associated with Down's syndrome that affects the transverse atlantal ligament that forms the posterior support of the odontoid process. The contribution to the instability by the abnormalities of the odontoid process that have also been described is less clear. An occasional association with an inflammatory process in the neck, such as pharyngeal infection or juvenile rheumatoid arthritis, has been noted. There is a preponderance of females with atlanto-axial instability in a ratio of more than 2:1. The reason for this is not known.

In Davidson's review, six individuals had post-traumatic

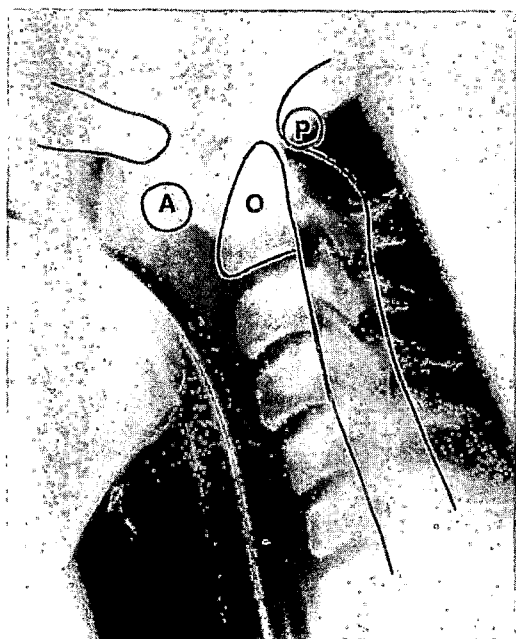


Fig. 1. Lateral radiograph of the cervical spine: O is the odontoid process of the axis, A the anterior arch of the atlas, and P the posterior arch. There is craniovertebral settling with atlanto axial subluxation.

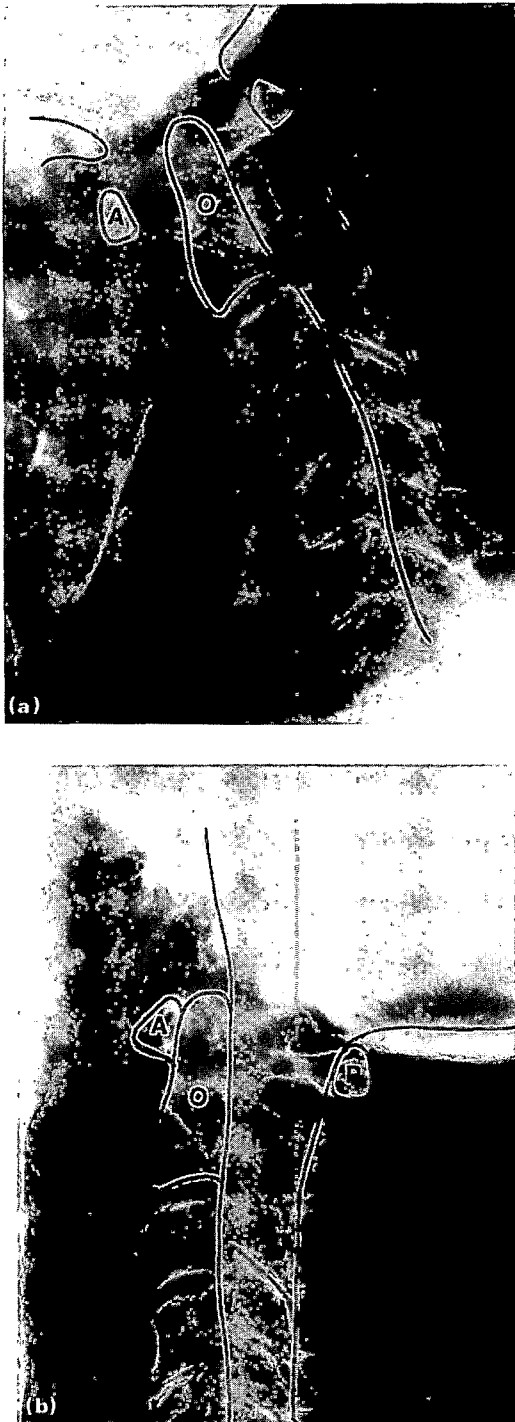


Fig. 2 (a) and (b). Lateral views of the cervical spine in flexion (a), and extension (b), of an adult with atlanto-axial instability. The flexion view shows narrowing of the spinal canal by the odontoid process (O) within the atlas (A-P).

dislocations, 15 presented with various combinations of neck pain, head tilt and torticollis, and 16 had an abnormal gait that included frequent falling or staggering that preceded the dislocation.⁵ Signs noted on examination included hyperreflexia, clonus, limb weakness and hemi-, para- or quadriplegia. He noted that in individuals with Down's who suffered dislocation, the event was preceded by neurological signs by between several weeks, months or even years.

Atlanto-axial subluxation related to anaesthesia had

been reported by Moore *et al.*⁷ Their patient was a 4-year-old boy with Down's syndrome who had a ventricular septal defect repaired apparently without problem. He was found to have reverted to crawling at postoperative review at one month, but had previously been able to walk upright. He was found to be paraplegic on examination. Computerised tomography showed posterior subluxation of the body of C₂ and the base of the odontoid process with spinal cord compression. Eight months after cervical fusion the patient had regained his previous gait. In another report, a 6-year-old boy with Down's developed severe right-sided neck pain one month after a successful repair of an atrioseptal defect.⁸ Motor and sensory examination was otherwise normal. Rotatory subluxation of the atlanto-axial joint was evident on conventional radiographs and on computerised tomography. C₁₋₂ fusion was performed and at follow-up the child was symptom free. Our patient's history of loss of locomotor skills, hypotonia and hyperreflexia fits the pattern of presentation of other Down's syndrome patients with symptomatic atlanto-axial dislocation.

The anaesthetist should pay particular attention to loss of locomotor skills or gait disturbance and to any complaint of neck pain in the pre-operative assessment of a child with Down's syndrome. The examination should note head tilt or torticollis and should exclude the presence of limb weakness and increased tone and reflexes that would suggest cervical spinal cord compression. Every Down's syndrome patient should have lateral radiographs of the cervical spine taken in flexion and extension to exclude asymptomatic atlanto-axial instability before elective general anaesthesia. Furthermore, any individual with Down's syndrome who has evidence either in the history or on examination of spinal cord compression requires urgent referral to a neurosurgical centre for consideration for spinal fusion. Every effort should be made to avoid all manoeuvres that involve forceful or excessive flexion, extension or rotation of the neck in asymptomatic individuals for whom radiographs are not available.

Acknowledgment

The authors thank Dr J. Herbetko (Senior Registrar, Department of Radiology, Southampton General Hospital) for his help in preparing the illustrations.

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32-gauge spinal catheters through 26-gauge needles

C. J. NAGLE, H. J. McQUAY AND C. J. GLYNN

Summary

Small diameter intrathecal catheters potentially combine the certainty of intrathecal injection and the advantage of repeatability without the risk of a high incidence of headache after dural puncture. We report problems placing such catheters.

Key words

Equipment; catheters, intrathecal.

Administration of drugs into the subarachnoid space has a number of advantages over the epidural route.^{1,2} The presence of cerebrospinal fluid (CSF) in the needle is a positive endpoint and leads to a lower failure rate in regional anaesthesia.^{2,3} Application of local anaesthetics as close as possible to their intended site of action means that the time until onset of effect is reduced substantially; this leads to quicker turnaround time in the anaesthetic room. The lower doses needed intrathecally reduce the risk of toxic systemic reactions to a minimum. Epidurals, on the other hand, have the advantage that the effect can be extended almost indefinitely by using a catheter. It is possible, by incremental dosing via a catheter, to control the extent of a block and to achieve a greater degree of cardiovascular stability because the slower onset allows time for physiological and pharmacological compensatory mechanisms to take place.²

The use of subarachnoid catheters is recognised to combine the advantages of an epidural with the benefits of a spinal.^{1,3,4} However, the high incidence of headache afterwards when large bore needles are used to introduce catheters has limited the usefulness of the technique.^{4,5} In principle therefore a spinal catheter with small outside diameter, enabling it to be introduced via a 25 or 26-gauge needle, offers considerable advantages both for intra-operative and long-term pain relieving manoeuvres. The recent availability of 32-gauge catheters has renewed interest in continuous subarachnoid techniques.⁶ We have used 20 of these catheters and encountered some difficulties in placing them, particularly in patients in whom the initial lumbar puncture was technically difficult.

Equipment

The MicroSpinal catheter (TFX Medical, High Wycombe, Bucks) is a 32-gauge polyimide catheter with a built-in high tensile stainless steel stylet that increases the strength and resists kinking of the catheter. They cost £52.48 each and will pass through most 25-gauge and many 26-gauge spinal needles; the small size of the needle should minimise the risk of headache after dural puncture.⁵ A lumbar puncture is performed and a threading aid is then attached to the hub of the spinal needle to centralise the MicroSpinal so that it passes smoothly from hub to shaft of the needle to place the catheter. The catheter is marked in centimetre increments to indicate when the tip of the catheter passes beyond the end of the needle to lie in the subarachnoid space. A resistance may be felt as the catheter tip turns the corner at the end of the needle. The catheter is then advanced further so that 1 or 2 cm lie in the subarachnoid space. The needle is then withdrawn over the catheter. A 1 ml Luer lock syringe and Tuohy Borst adaptor are supplied; these are necessary because of the high pressure required to inject drug through a tube 91-cm long with a cross section area of only 0.0002 sq cm.

We have often encountered problems despite the apparent simplicity of the technique.

Problems in feeding the catheter

It is almost impossible to occlude the lumen of the catheter, (it is still possible to inject when a knot is tied in it), but relatively small forces applied longitudinally cause the catheter to kink because the stylet bends. Such kinking does not

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Accepted 4 May 1990.

affect the ability to inject, but even a minimal degree of bend makes it difficult to thread the catheter further and impossible to retract the needle over the catheter. It is necessary, when this happens, to remove the catheter and needle together and start again because of the risk of shearing the catheter on the sharp bevel of the needle. This second dural tap increases the chance of a headache and doubles the cost since a new catheter set has to be used because at present the catheters are only marked at one end.

Kinking occurred at two stages during insertion; as the tip of the catheter turned the corner at the end of the needle and when resistance was felt with 1–2 cm of the catheter in the subarachnoid space. Resistance with 2 cm of catheter beyond the tip of the needle was a repeated and repeatable phenomenon. We think it occurred because the catheter snagged on a nerve root or hit the dura mater anteriorly. Attempts to recreate the situation in the postmortem room, where we could have dissected out the cord to find the cause of the resistance, were unsuccessful—it was not possible to introduce the catheter in any of four corpses. The manufacturers recommend that no more than 2 cm be introduced into the subarachnoid space for fear of knotting the catheter. Our experience with long-term epidural catheters suggests that considerable loss of catheter length may occur in ambulant patients because of repeated flexion and extension of the back. With only 1 or 2 cm of spinal catheter in the subarachnoid space at the outset, there is no provision for loss of length through patient movement in long-term use.

Great care is needed to prevent pulling the catheter out with the needle during removal of the spinal needle after successful placement of the catheter. Again, if there is only one cm in the space to begin with, the margin of error is very limited.

Problems in feeding the catheter may be minimised by holding it as close to the hub of the needle as possible. It is also better to make small, repeated movements rather than one continuous one, and to avoid the temptation to overcome resistance by increasing the force on the catheter, since that leads to its kinking. Extreme vigilance while removing the needle is also needed. Difficulties in manoeuvring the catheter beyond the needle tip and into the subarachnoid space may be overcome by rotation of the needle or advancing or withdrawing it slightly.

Care needs to be taken when connecting the catheter to the Tuohy Borst adaptor. If too much catheter is threaded into the adaptor, the attachment of a syringe may kink the catheter and prevent injection. Too little catheter threaded allows the rubber cushion in the adaptor to occlude the end of the catheter. The adaptor must be screwed tight because the high pressure needed to inject through such a small catheter results in a substantial loss of drug through any leak around the adaptor. It is necessary to use a syringe with a Luer lock because the pressure generated is likely to cause disconnection of a push fit joint between syringe and adaptor. The mechanical advantage of a 1-ml syringe helps to produce enough pressure to inject through the tiny catheter. We found it an advantage to inject without holding the adaptor, since a finger around it can obscure a leak from the sealing cushion.

Problems in retracting the needle over the catheter

We have also had problems on two occasions after the

catheter was positioned in the CSF. On the first, blood was seen in the hub of a 25-gauge Becton Dickinson (BD) 8.75 cm spinal needle during the course of insertion. The needle was removed, redirected and inserted to give a clear CSF tap. In the second case lumbar puncture was very difficult, but eventually slightly blood-stained CSF was seen. In both cases 32-gauge MicroSpinal catheters were threaded to give 2 cm and 3.5 cm in the subarachnoid space. We then attempted to retract the needle over the catheter. Each time the catheter kinked at the hub of the needle. When the needle and catheter were removed together from the patients, it was possible to pull the catheter through the needle but impossible to retract the needle over the catheter. Blood clots were seen when the needles were flushed with saline. The catheters moved easily in either direction through the spinal needle after the saline flush. It seems that a small amount of blood in the spinal needle may prevent the needle being withdrawn over the catheter, even though the catheter was threaded through the needle without difficulty.

We performed a bench test. First, blood was injected into a spinal needle and left for 2 minutes, after which a catheter would not thread through it. The blood could be cleared from the needle with difficulty using saline. Second, blood was introduced into a clean needle and a catheter introduced immediately; it threaded without difficulty, but 2 minutes later it was not possible to advance the catheter further, although the catheter could be pulled back through the needle.

It is necessary, when placing a catheter in a patient, to retract the spinal needle over the catheter, which is equivalent to advancing the catheter. Pulling the catheter back through the needle is contraindicated because of the likelihood of it shearing on the bevel of the needle. TFX state in the product information that their catheters will pass through 25- or 26-gauge BD needles. The internal diameter of both these needles is 0.25–0.3 mm. The external diameter of the 32-gauge catheter is 0.24 mm. Thus the clearance between the catheter and the inside of a needle may be as little as 10 μm . Even a trace of blood is likely to cause difficulties during placement of a catheter.

We recommend therefore that if blood enters the spinal needle at any stage of insertion of a MicroSpinal catheter, either through passage through a superficial vein or through a bloody CSF tap, that attempt should be abandoned, and the procedure should be restarted using another spinal needle.

Conclusion

Spinal catheters small enough to be introduced through a 26-gauge needle have renewed interest in continuous spinal analgesia. They should avoid the major drawback of a high incidence of headache after dural puncture. It remains to be seen whether difficulties in placement of these catheters limit their clinical usefulness.

Acknowledgments

We thank Mr T. McGuerk of TFX Medical, High Wycombe, Buckinghamshire for his help and for supplying the MicroSpinal catheters.

Pressure generated during insertion of lumbar epidurals

A comparison with the Portex epidural injection simulator

J. A. LANGTON AND B. H. MEIKLEJOHN

Summary

The aim of this study was to measure pressures exerted during insertion of lumbar epidurals in obstetric patients. The pressures were compared with those obtained when the Portex epidural injection simulator was used, and were significantly greater when a saline technique was used compared to those when air was used ($p < 0.05$). There were no differences between the pressures obtained with the simulator and those measured *in vivo*.

Key words

Anaesthetic techniques, regional; epidural.
Equipment; simulator.

Possible differences in the incidence of complications during location of the epidural space prompted an investigation into the pressures generated when two different loss-of-resistance techniques, air or saline, were used to locate the lumbar epidural space. The Portex epidural injection simulator was recently described,¹ and we compared the pressures generated *in vivo* with those generated when the simulator was used.

Method

Twelve women who requested epidural analgesia during labour were studied after Ethics Committee approval and informed consent were obtained. They were allocated randomly to either the air or saline group, and a lumbar epidural was inserted using the loss-of-resistance technique with a 'Steriseal' 16-gauge Tuohy needle. The pressure generated during location of the epidural space was measured using a three-way tap interposed between the syringe and the hub of the Tuohy needle. The tap was connected by a 1-m length of manometer tubing to a Honeywell piezoresistive pressure transducer, which was calibrated according to the manufacturer's instructions, and the output connected to a BTI Biox 2100 chart recorder.

Insertion pressures were measured in a similar manner in the second part of the study using the Portex epidural injection simulator. Six anaesthetists of varying experience, from consultant to senior house officer, identified the 'epidural space' using air first, then saline.

The results were analysed using a two sample *t*-test. Significance was taken to be a *p* value < 0.05 .

Results

The pressures generated *in vivo* when the two different loss-of-resistance techniques for location of the epidural space were used were different. (Fig. 1). In the air group, the mean pressure was 115 mmHg (SD 49.03) and in the saline group 1017 mmHg (SD 336.4) ($p < 0.05$).

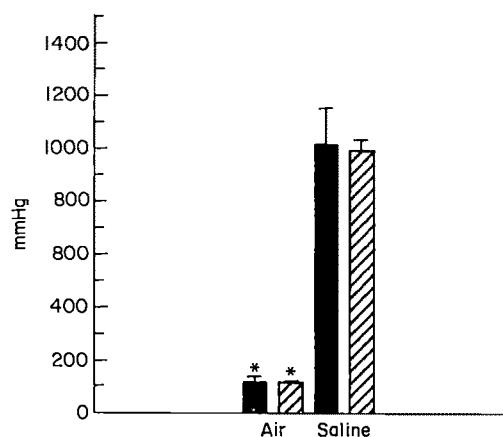


Fig. 1. Pressures (mmHg) exerted during insertion of lumbar epidurals (* $p < 0.05$). ■, patient pressures; ▨, simulator pressures.

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Accepted 9 May 1990.

The pressures generated when using the Portex epidural injection simulator were comparable to those in the clinical setting. Loss of resistance using air produced a mean pressure of 112 mmHg (SD 23.92) while a mean pressure of 992 mmHg (SD 178.4) was generated with saline (Fig. 1). There was no significant difference between the *in vivo* pressures and those using the simulator.

Discussion

Previous studies noted that the pressure generated when air is used is greater than 200 mmHg;² other workers commented on the considerable force required to advance the Tuohy needle through the tough ligamentum flavum.³ We demonstrated in this study a difference between the pressures generated when using loss of resistance to air or saline to locate the epidural space. We also showed that the Portex epidural injection simulator accurately reproduces the pressures encountered in clinical practice; this confirms its value as a teaching aid.

It has been reported⁴ that there are differences in the dural puncture rate when using loss of resistance to air or saline to locate the epidural space, with a lower incidence using the saline technique. It may be that the greater pressures generated by the use of saline make blockage of the Tuohy needle by ligamentous tissue less likely to occur and this may be a factor in the lower dural puncture rate of the saline technique.

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Antireflux valves in patient-controlled analgesia

M. T. KLUGER AND H. OWEN

Summary

Antireflux valves are widely used in conjunction with patient-controlled analgesia devices. It is important to appreciate the limitations and dangers of these systems. They can achieve a potential 'stored volume' if occluded and they may, as part of the administration set, retard fluid administration. Seven antireflux systems currently available were tested in conjunction with three patient-controlled analgesia pumps. The systems' volume, time to occlusion alarm and flow rates were measured. Results showed that the sets with low stored volumes were less efficient as administration sets. A potentially dangerous bolus could result after release of occlusion if sets with large stored volumes were used in conjunction with pumps that utilised concentrated solutions of opioid. This study has identified the ideal antireflux valve system.

Key words

Equipment; infusion systems, patient-controlled.

The need for antireflux valves during patient-controlled analgesia (PCA) was recently highlighted by the Emergency Care Research Institute (ECRI).¹ The valves are necessary to prevent retrograde pumping of analgesic agent into the tubing of a parallel, gravity-driven infusion should the intravenous cannula become obstructed. The feedback loop is opened if this happens, the technique fails and a dangerous dose of drug could be delivered as a bolus when the blockage is cleared to restart the infusion.

The ECRI recommendation for PCA systems is that if complete occlusion occurs the pump should alarm within three dose activations at every dose setting. The pressure in the system required to activate the occlusion alarm is determined by the manufacturer; it is generally high (over 300 mmHg) to overcome the internal friction of the syringe and to accommodate the pressure required to overcome the flow restriction of small-bore cannulae and delivery tubing. The effect of adding an antireflux valve to the drug delivery system has not been studied.

The volume of drug solution expelled from the PCA device and delivery tubing after releasing an occlusion (which we have called the stored volume) is determined by the setting of the occlusion pressure alarm and the compliance of the system (tubing plus valve). One study reported that surges of up to 0.5 ml can be delivered from an infusion pump connected to standard tubing after release of an obstruction.² The number of milligrams of

opioid contained in this stored volume is then a product of the concentration of the solution.

Some manufacturers of PCA devices also produce dedicated giving-sets for their equipment, whereas others either suggest or recommend a particular product. One PCA device is in widespread use at this hospital and two others were considered and used with several antireflux systems.

It was clear from discussion with colleagues using PCA, and some equipment suppliers, that the clinical relevance of 'stored volume' had largely been ignored and so was worth further investigation. It was decided formally to compare some PCA and antireflux systems, paying particular attention to the 'stored volume'. Flow-rate through the antireflux valves and the potential for misconnexion were also studied.

Methods

The characteristics of seven different antireflux systems (Fig. 1), when used with three PCA pumps, were investigated. The PCAS (Graseby, Watford, UK) and MDS 110 (Bionica, Castle Hill, NSW) were tested with the following antireflux devices (see Appendix for list of manufacturers): Tuta in two configurations; Lifemed, Cardiff (v.y.c. Con), Abbott PCA, Abbott PCA Mini Bore with antisiphon (Abbott 2) and Quest Medical. The Lifecare 4100 (Abbott, North Chicago) was tested only in conjunction with its own dedicated giving set for comparison.

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Accepted 15 February 1990.

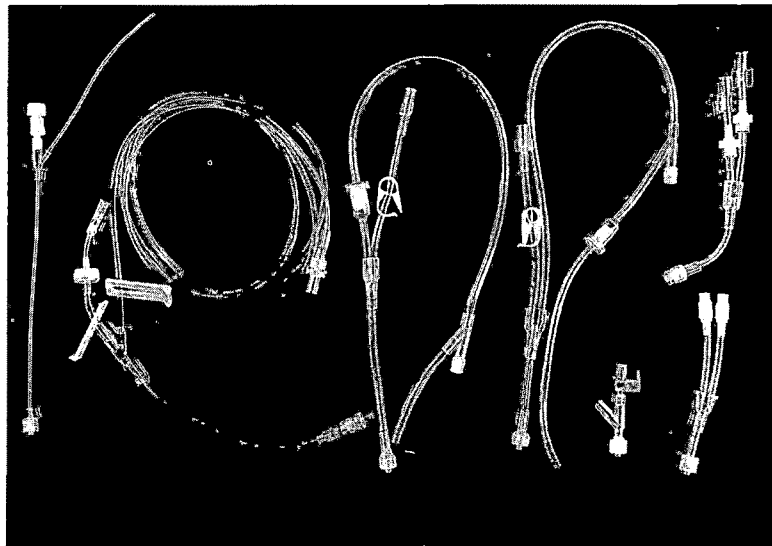


Fig. 1. Antireflux valves used; left to right; Abbott 1, Abbott 2, Tuta 1, Tuta 2, Cardiff, Lifemed (Above) and Quest (Below).

Stored volume

The pumps were set to deliver the maximum bolus dose at their highest rate of delivery. Each pump was tested in turn four times with each of the seven antireflux valves, using either the integral part of the antireflux valve (Tuta 1 and 2) or a standard Tuta low compliance tubing (length 75 cm, deadspace 1.25 ml, part no. 50-308) in a configuration used clinically. Each administration set had a three-way valve at its termination which was used to create an occlusion. The pumps were primed with 0.9% saline and activated to deliver a bolus with the three-way tap in the closed position. The time taken for the occlusion alarm to sound was measured using an analogue stopwatch. When the occlusion alarm sounded, it was silenced and the tap opened. The fluid expelled from the tubing ('stored volume') was collected in a preweighed beaker on a calibrated top-loading electric balance (Model PL 1200, Mettler Instruments, AG, Zurich, Switzerland). The volume was determined from the mass of fluid and the specific gravity of 0.9% saline at ambient temperature (1.006 at 22–25°C). The time to postocclusion alarm and the 'stored volume' released were recorded for all combinations of the antireflux valves with the Graseby and Bionica pumps. The Abbott was tested solely with its dedicated giving set. A new antireflux device was used for each determination, to prevent bias from any hysteresis in compliance, which may occur with cyclical pressurisation of the set.

Retardation of fluid administration

A protocol, similar to that used to investigate the efficiency of the Cardiff valve, was carried out in order to assess the adequacy of the valves as part of the administration set.³ Each valve system was connected to the Luer connector of a standard intravenous infusion giving set primed with 0.9% saline. The drip-controller was turned on and the time for 200 ml of saline to flow into a measuring cylinder was measured when the surface of the saline was 50, 100 and 150 cm above the outlet of the one-way valve system.

This provided a range of pressures from 5–15 kPa, which corresponds to that found in normal clinical practice, between the reservoir and the patient's venous system. The measurements were repeated three times at each height, and also without any valve present.

Results

The time to alarm with the PCAS (Fig. 2) ranged from 50 seconds (Abbott 2) to 105 seconds (Quest). The stored volume expelled after release of occlusion ranged from 1.03 ml (Abbott) to 2.06 ml (Tuta 2). The MDS 110 (Fig. 3) had

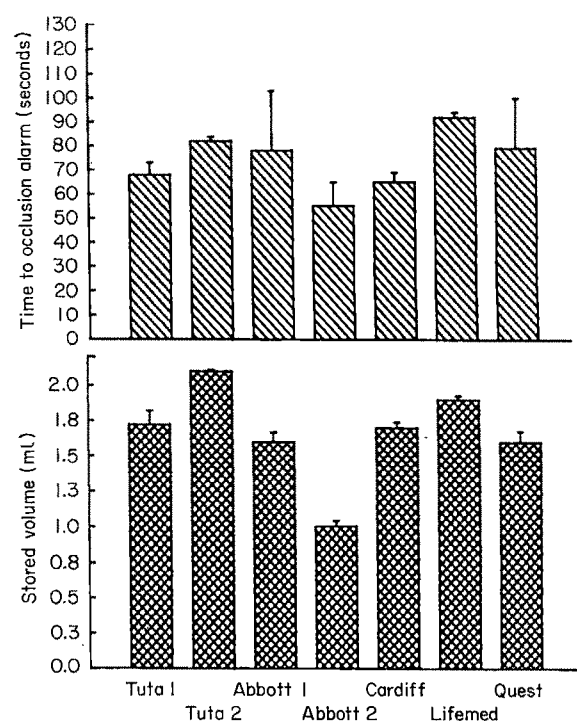


Fig. 2. Time to occlusion alarm and stored volume with the Graseby PCAS. $n = 4$ for each group.

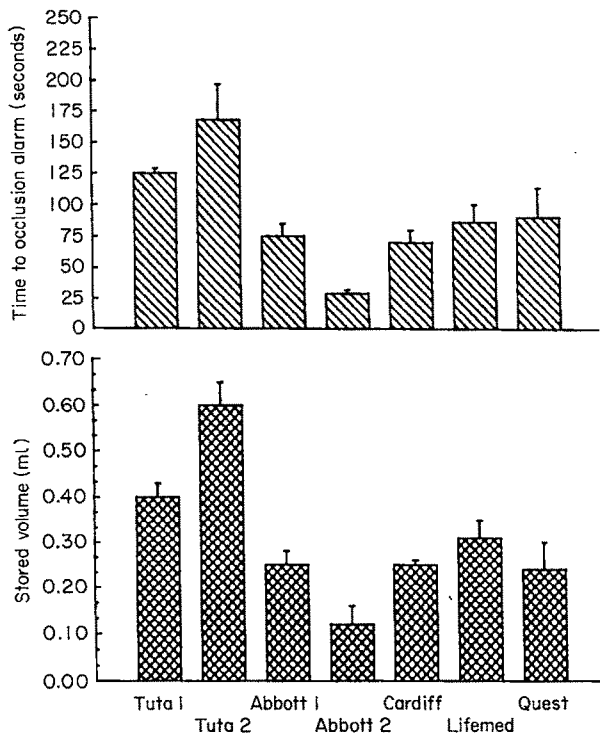


Fig. 3. Time to occlusion alarm and stored volume with the Bionica MDS 110. $n = 4$ for each group.

wider range of occlusion times, 24 seconds (Abbott 2) to 210 seconds (Tuta 2). The volumes associated with this pump were much smaller, 0.08 ml (Abbott) to 0.6 ml Tuta 2. The Lifecare 4100 with its special set had a mean time to occlusion of 59.5 seconds and stored volume of 1.24 ml.

The results of the flow studies (Fig. 4) show that the most efficient administration set, as demonstrated by the most rapid flow rate, were the two Tuta sets. These proved to have a rate of delivery equal to the control drip set with no antireflux valve present. The Abbott PCA set reduced the

maximum flow rate to one third of the more efficient sets and although the newer Abbott 2 was more efficient at higher pressures, it was not an improvement over the PCA or other sets at lower pressures. The Cardiff, Quest and Lifemed were intermediate in their performance.

Discussion

The effectiveness of antireflux valves was tested to find out whether their intrinsic compliance and possible 'stored volume' led to the delivery of a bolus of drug when an occlusion was released. We found that the Tuta sets, with their wide-bore tubing and large, floppy, compliant valve leaflets, have consistently higher stored volumes than the other antireflux devices. This becomes clinically relevant in the context of PCA and the pump used in conjunction with the antireflux device. We routinely use morphine sulphate at a concentration of 1 mg/ml. Under these conditions in the Graseby PCAS, an accidental dose of approximately 2 mg could be administered after an occlusion was cleared. This is within an acceptable dose range, although undesirable. It should be noted that this pump can be programmed to provide a concentration range from 10 $\mu\text{g/ml}$ to 99.5 mg/ml, so more concentrated solutions could be used. However, the Bionica MDS-110 PCA has a minimum concentration setting of 10 mg/ml and so would have the potential of administering a 6-mg dose of opioid. This could have potentially serious effects and would be unacceptable in clinical practice. The bolus administered is still significant, even in conjunction with other valves such as the Cardiff, Lifemed and Abbott; 2.5 mg, 3.1 mg and 2.5 mg respectively. The differences in time to occlusion alarm, although longer with the antireflux devices used in conjunction with the Bionica pump, were clinically insignificant.

It was also questioned whether the addition of one of the valves hindered intravenous fluid administration. It seems from the results that the most effective antireflux valves, that is with respect to low stored volume and short occlu-

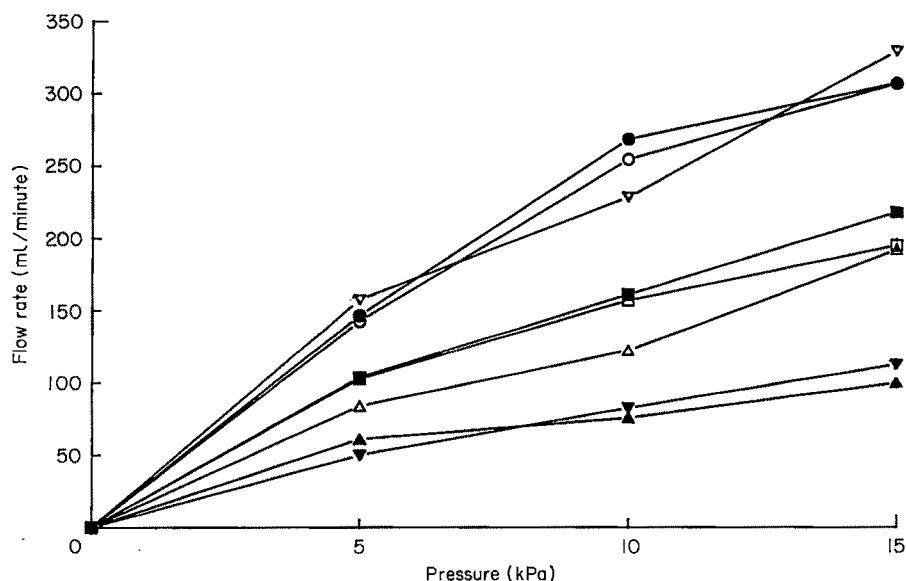


Fig. 4. Flow rate characteristics of the seven antireflux devices when tested at three different pressures. ○—○, Tuta 1; ●—●, Tuta 2; △—△, Cardiff; ▲—▲, Abbott 1; ▼—▼, Abbott 2; ■—■, Lifemed; □—□, Quest; ▽—▽, no valve.

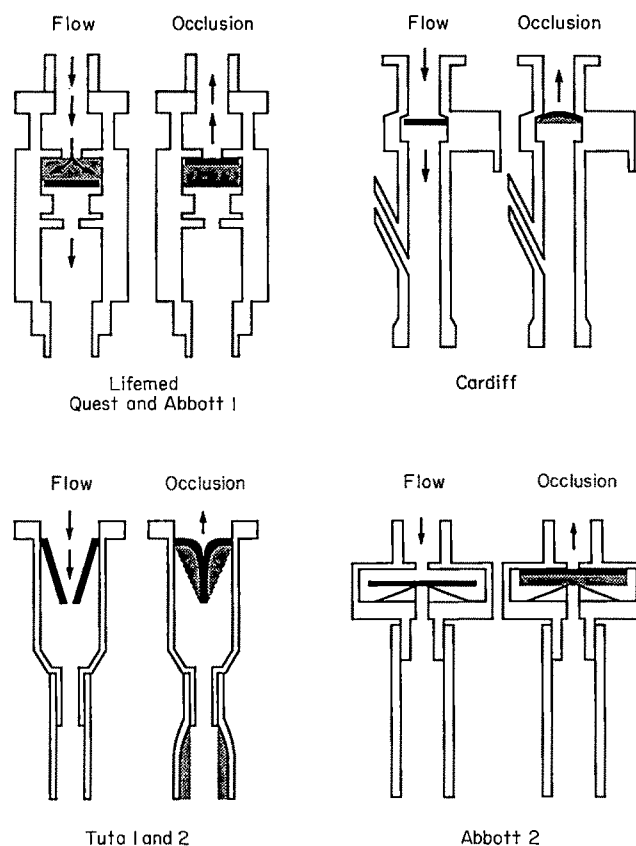


Fig. 5. Schematic illustration (not to scale) of the antireflux valves used in the study. Shaded areas correspond to stored volume.

sion alarm times, in particular the Abbott sets, significantly hinder the maximum flow rates associated with fluid administration. Conversely, the sets with the potential for large stored volumes, for example the Tuta sets, are able to deliver fluids as rapidly as the normal drip sets.

The stored volume has two components, the compliance of the tubing and deformability of the valve. Most of the antireflux valves are small flap valves, but the prototype Tuta sets have large duck-billed valves with large valve leaflets which during occlusion testing can be seen to invert, but under normal conditions do not retard rapid flow (Fig. 5).

The flap-valve in the original Cardiff valve could be rotated so that in one position it did not function as an antireflux device, whereas at another setting it completely occluded flow. The potential for misuse is immediately apparent and the version of Cardiff valve we tested does not have this facility. The infusion can still be incorrectly connected to devices such as the Cardiff valve, Abbott PCA and Abbott 2 sets, since there is only one valve present on a two-limbed infusion system.⁴ This again may defeat the purpose of using these systems. The Tuta sets do not have this potential problem, because they have only one access port. Both the Lifemed and Quest products have valves on both limbs so that identification of the protected limb is unnecessary. This is especially useful when PCA is first introduced and personnel are inexperienced in its use.

The Lifemed device is sterilised by ethylene oxide and therefore the protecting plugs at the ends of each limb have a central hole. Thus, if only one limb is to be used, for example during initiation of intravenous therapy before

induction of anaesthesia, the end plug needs to be replaced. The systems sterilised by gamma-irradiation do not have this irritating problem. An antireflux valve in place intra-operatively prevents blood being forced backwards along the infusion line by a noninvasive blood pressure monitor on the same arm.

The technical specifications of modern PCA pumps are increasingly complex to ensure greater accuracy and safety. However the patient/machine delivery interface, that is the connecting set and antireflux device, was shown to have significant effects on the function of the overall PCA system. The disposables must be considered as important as the pumps themselves for the safe administration of PCA, and the manufacturers should take account of this. Moreover, when in use with pumps that rely on small volumes of concentrated solutions, for example the MDS-110 PCA, they can present a potential problem. It was previously shown that occlusion of syringe pumps can generate substantial in-line pressures that range from 250–1600 mmHg and deliver a bolus of 0.5 ml;² we have shown similar results in the context of PCA machines and antireflux devices. We suggest that, for safety, dilute solutions be used in PCA systems for postoperative pain control. This might avoid recurrence of some of the accidents that have been reported with PCA.⁵ Nursing staff need to be informed of these dangers and, in particular, must ensure that if the occlusion alarm has been activated the system is disconnected before the line is unblocked.

PCA requires a drug-delivery system, designed to prevent incorrect configuration, with low intrinsic compliance and a wide bore to allow rapid fluid administration. It is also

suggested that the ideal system should be sterilised by irradiation and be inexpensive. Currently, if the antireflux valve system is to be incorporated in the intravenous line, for example, to facilitate drug infusion during surgery, then the Tuta sets will not retard fluid administration. However, the Abbott sets would be safest if the system is added after operation specifically for PCA administration. It appears that the ideal system is still to be marketed.

Acknowledgments

The authors are particularly grateful to E. Loveday of Abbott Australasia, T. Sofu of Tuta Laboratories, G. Middleton of Implox for providing samples of their products and Dr J. Plummer for reviewing the manuscript.

Note added at proof

The Bionica pump has since been modified so that drug dilutions down to 1 mg/ml can be programmed.

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Appendix

List of manufacturers of antireflux valves

Tuta, Lane Cove, New South Wales, Australia.

Lifemed, Biospectrum, Chatswood, New South Wales, Australia.

Cardiff, Vygon, Uxbridge, UK.

Abbott PCA Set, Abbott Laboratories, N. Chicago, Illinois, USA.

Quest, Quest Medical, Carolton, Texas, USA.

The Ohmeda 9000 syringe pump

The first of a new generation of syringe drivers

D. N. STOKES, J. E. PEACOCK, R. LEWIS AND P. HUTTON

Summary

The Ohmeda 9000 syringe pump was developed in response to the need for an infusion apparatus to administer intravenous anaesthetic agents. It incorporates a bolus facility for the rapid, controlled delivery of loading infusions, or incremental dosing over a background maintenance infusion, and may be interfaced with a controller for computer-driven infusions. The Ohmeda 9000 pump has undergone bench testing and detailed evaluation in clinical practice using a variety of syringe sizes and makes. It is capable of accurate delivery over a range of infusion rates provided the recommended manufacturers' syringes are used with the appropriate pump setting. The pump was easy to use and reliable in clinical research and routine clinical practice. It should find its niche as the first genuinely 'anaesthetist-friendly' infusion pump.

Key words

Equipment; syringe pump.

The introduction of new anaesthetic agents with suitable pharmacokinetic properties for administration by continuous intravenous infusion (e.g. propofol) has resulted in the need for an infusion apparatus specifically designed for the anaesthetist who wishes to practise total intravenous anaesthesia. This has stimulated new developments in infusion pump philosophy and technology. The Ohmeda 9000 syringe pump (Fig. 1) was evolved to meet the everyday requirements of the clinical anaesthetist using infusional anaesthesia.

Description

The Ohmeda 9000 is a syringe driver powered by an electric motor which drives a leadscrew transmission system. It possesses a snap-in syringe cradle capable of recognising 10-, 20- and 50- or 60-ml syringes by their external diameters from any of three preselected manufacturers (BD-Plastipak, Terumo and Sherwood-Monoject). The snap-in mechanism is designed to facilitate insertion and removal of syringes with one hand only.

The pump is controlled by six touch key pads and a rotary control dial. A liquid crystal display (LCD) indicates the syringe size, infusion rate and volume infused, together with other features described below. A status light-emitting diode (LED) indicates standby (continuous green), running

(green flashing), alert (amber flashing) or alarm states (red flashing).

A check sequence is activated by pressing the on-off touch key. The preselected syringe type is displayed by the pump during its warm-up sequence (SH—Sherwood; bd—Plastipak; tE—Terumo). It can be altered by simultaneously pressing the on-off and alarm-mute touch keys then rotating the dial until the required make is displayed. The pump should then be turned off again before subsequent use. The desired infusion rate can be set by rotation of the dial after pressing the rate key. The rate increment is proportional to the speed at which the dial is rotated. Delivery rate can be specified to 0.1 ml/hour up to 10 ml/hour, and 1.0 ml/hour thereafter up to a maximum rate of 200 ml/hour. The stop-start touch key is used to control the delivery of the infusion. Pressing the bolus touch key once displays one of three rapid infusion rates that can be selected using the rotary dial; further sustained pressure on the touch key within 5 seconds starts a fast infusion at the preselected rate. Bolus delivery is sustained as long as pressure is maintained on the touch key, and the bolus volume infused is displayed continuously by the LCD. This volume is subsequently added on to the LCD display of total volume infused by the pump. Bolus infusion rates of 300, 600 or 1200 ml/hour may be selected for 50- or 60-ml syringes; 300, 350 or 450 ml/hour for 20-ml

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Accepted 23 January 1990.

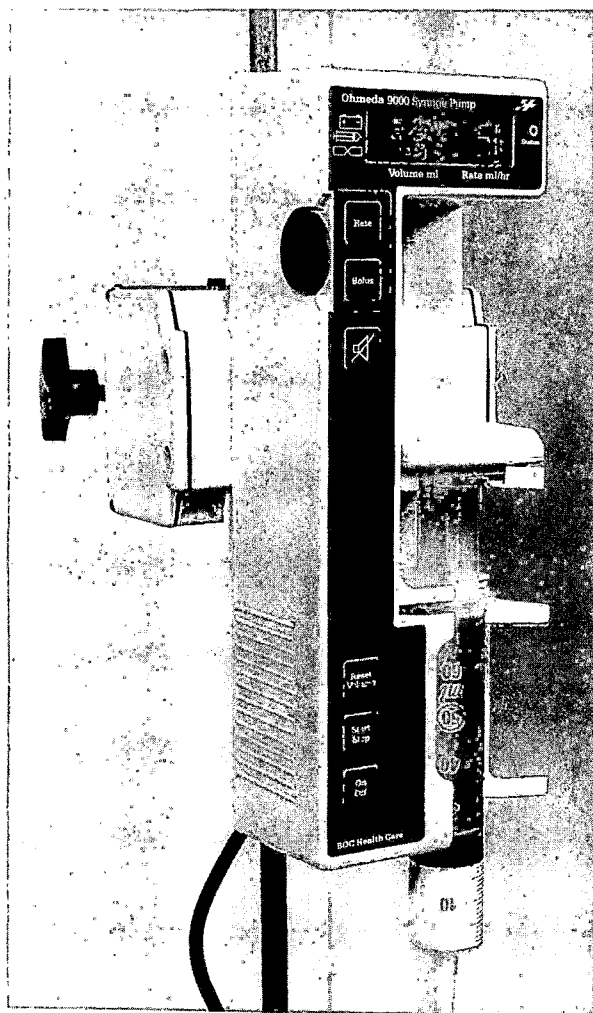


Fig. 1. The Ohmeda 9000 syringe pump.

syringes, and 180, 220 or 300 ml/hour for 10-ml syringes. Pressing the volume reset touch key returns the volume display to zero, if the rapid infusion facility is used to prime the infusion line.

Alarms for wrong syringe, syringe empty, battery discharged and high line pressure or occlusion are indicated by alert flags on the LCD and accompanied by audio tones, status LED flashing red and discontinuation of the infusion. The alarm cancel touch key disables the audio tone for 30 seconds. A lower level of alert conditions are activated when the battery is close to discharge or the syringe volume is less than 10% of capacity. The high line pressure alarm is set to activate if the line pressure exceeds 700 mmHg during continuous infusion, or 1200 mmHg during bolus delivery.

In the free-standing, battery-operated mode the Ohmeda 9000 pump should continue to operate for 8 hours. The mains-powered backbar engages the pump in a vertically mounted tongue-and-groove connexion and can be clamped to any vertical pole or located on the anaesthetic machine. The backbar contains a step-down transformer, and electrical contact with the correctly located pump is through matching pairs of brass connectors. This permits mains operation of the pump and simultaneous recharging of the batteries. An optional RS232C interface may be

located on the backbar for the delivery of computer-controlled infusions.

Methods

A prototype of the Ohmeda 9000 syringe driver indistinguishable from the final model in electrical and mechanical construction was evaluated. A series of bench tests were performed to assess accuracy of delivery of infusate from a variety of syringe makes and sizes. Clinical studies were performed in which neuromuscular blocking agents, analgesics and sedatives were infused in both operating theatre and intensive care environments.

Bench tests

The Ohmeda 9000 syringe pump was instructed to deliver infusions of 0.9% saline solution or time-expired packed red blood cells at rates of 2, 5, 40 or 90 ml/hour from 10-, 20- or 60-ml syringes manufactured by Becton Dickinson (BD Plastipak) or Sherwood (Monoject). The infusions were delivered through a 200-cm long, 1-mm internal diameter catheter (Lectrocath: Vygon) which was primed before commencing each run. The volume infused over each 10-minute interval was collected into a preweighed beaker one minute after activating the pump. Infusion volumes were derived gravimetrically using a Mettler balance (Gallenkamp) after correcting for the density of the infusate. Ten measurements were made at each infusion rate with each syringe. Ten-millilitre syringes were not tested at 90 ml/hour because of their smaller capacity. A comparison was made with syringes manufactured by Braun (Omnifix) which are not recommended for use with the Ohmeda pump. The internal and external diameters of all syringes were measured.

Clinical evaluation

The Ohmeda 9000 syringe pump in a preliminary study, was used to infuse either propofol (Diprivan: ICI Pharmaceuticals), midazolam (Hypnovel: Roche) diluted to 1 mg/ml, atracurium (Tracrium: Burroughs Wellcome), alfentanil (Rapifen: Janssen) or papaveretum (Omnopon: Roche) diluted to 1 mg/ml. The infusions were administered from 10-, 20- or 60-ml BD Plastipak (Becton Dickinson) syringe through a 200-cm long, 1-mm internal diameter catheter (Lectrocath: Vygon) into peripheral or centrally located intravenous cannulae. The drugs were administered as clinically indicated in patients undergoing major surgery, or intermittent positive pressure ventilation on the Intensive Care Unit. The duration and volume of drug delivered for loading and maintenance infusions was recorded in each case. Subjective assessments of various aspects of pump performance and 'user friendliness' were made.

In a subsequent study the Ohmeda 9000 syringe pump was used to infuse propofol for the induction and maintenance of anaesthesia supplemented by opioids and nitrous oxide in 180 patients undergoing peripheral body surgery. Propofol was infused at 5, 10 or 20 ml/minute during induction of anaesthesia, and subsequently at 6 (mg/kg)/hour supplemented with bolus doses as required.

Table 1. Percentage errors in volumes of normal saline infused and coefficients of variation.

Syringe make (pump setting)		% error at stated infusion rate (coefficient of variation)			
	Size (ml)	2 ml/hour	5 ml/hour	40 ml/hour	90 ml/hour
BD Plastipak (bd pre-set)	50	1.14 (0.12)	-3.6 (0.036)	-0.94 (0.008)	-1.06 (0.006)
	20	-4.53 (0.044)	-2.65 (0.035)	-1.07 (0.004)	-0.05 (0.004)
	10	-11.3 (0.091)	-4.84 (0.033)	-3.39 (0.007)	—
Sherwood Monoject (SH pre-set)	20	-0.54 (0.048)	-2.3 (0.018)	-1.17 (0.007)	-0.45 (0.004)
	10	-2.34 (0.049)	-2.21 (0.023)	-1.55 (0.004)	—
Braun* Omnifix (bd pre-set)	30	32.61 (0.084)	31.17 (0.023)	32.94 (0.008)	33.38 (0.003)

*Not recommended for use with the Ohmeda 9000 pump.

Results

Bench tests

The Ohmeda 9000 pump consistently delivered volumes of saline and blood accurate to within plus or minus 5% of the volume predicted, when the correct syringe type was preselected; the only exception was an 11% error because of underdelivery when the 10-ml BD-Plastipak syringe was set at 2 ml/hour (Table 1). These results were considered to be acceptable for clinical practice.

However, when an incorrect syringe type was preselected, greater inaccuracies in volume delivery were encountered. Sherwood Monoject and Braun Omnifix syringes overinfused by up to 18% when the pump was preset for BD-Plastipak syringes. Furthermore, a 30-ml Braun Omnifix syringe was recognised by the pump as a 20-ml syringe, which caused infusion of volumes 30% greater than predicted. These inaccuracies can be related to differences in diameters of the barrels of syringe used (Table 2).

Infusions with 10-ml size syringes of all makes tested were frequently interrupted by activation of the occlusion alarm. This appeared to be the result of 'sticking' of the plunger against the inner wall of the syringe barrel ('stiction'), and irregular motion of the syringe plunger was observed during infusion in some cases. The resistance was usually overcome by delivering a small bolus using the rapid infusion facility. Forty per cent of normal saline infusions at 2 ml/hour using the recommended syringe types and pump settings were interrupted. Any infusion interrupted by an occlusion alarm was abandoned and repeated. 'Stiction' could result in underdelivery at low infusion rates, as observed with the 10-ml BD-Plastipak syringe operating at 2 ml/hour (Table 1). No infusions of

packed red cells were possible using 10-ml syringes because of repeated activation of the occlusion alarm. Increased resistance to flow from the high viscosity of the infusate, together with 'stiction', probably account for this. Infusions of packed red cells using 20- and 60-ml syringes were accurate to within 2%.

The occlusion alarm in the continuous infusion mode was activated when line pressure exceeded 700 mmHg as measured by a Bourdon gauge; this is in agreement with the manufacturer's specifications.

In addition to the mean percentage error, the reproducibility of any setting is also very important. This was measured by calculating the coefficient of variation¹ on the raw data for each set of results (Table 1). The consistently low values indicate a high degree of precision in delivery by the Ohmeda 9000 pump.

Clinical evaluation

Ninety-nine infusions were delivered during the preliminary study consisting of propofol (10), midazolam (18), atracurium (22), alfentanil (25) and papaveretum (24). The mean duration of infusion was 255 minutes and the mean volume infused was 23.2 ml. The rapid infusion facility was used to deliver a loading infusion in over half of the patients and increments of propofol to deepen anaesthesia on 11 occasions. Satisfactory drug delivery was achieved in all patients except one who received atracurium from a 10-ml syringe. The occlusion alarm activated repeatedly although the delivery line was patent and not kinked. Delivery of a bolus of 0.5 ml was effective in cancelling the alarm on each occasion. It seems likely that the interruptions to flow were caused by 'stiction'.

The overall 'user friendliness' of the Ohmeda 9000 pump was confirmed by subjective practical assessments. Ease of set-up and adjustment of infusion and bolus rates, performance of alarm systems and mechanical reliability were entirely satisfactory. However, syringe insertion and replacement were judged to be unsatisfactory in 32 and 18% of cases respectively. The snap-fit retainer for the syringe plunger located on the carriage mechanism was unduly stiff and made the insertion and replacement of syringes with one hand impossible. In 5% of cases the force required to complete these manoeuvres was sufficient to cause loosening of the backbar clamp on the drip stand. This problem was mainly encountered at the start of the study and a learning effect was observed as syringe insertion technique was modified. It is unlikely that the preci-

Table 2. Internal and external diameters of syringes.

Syringe make	Size (ml)	Inner diameter (cm)	Outer diameter (cm)
BD-Plastipak	50	2.62	2.87
	20	1.91	2.15
	10	1.42	1.62
Sherwood Monoject	20	2.04	2.27
	10	1.59	1.80
Braun Omnifix	30	2.28	2.34
	20	2.01	2.18
	10	1.60	1.77

Table 3. Desirable properties of an infusion device for anaesthesia.

1. Reliable and electrically safe.
2. Accurate and consistent delivery at rates between 0.1 and 200 ml/hour.
3. Practically easy to set up and use.
4. Portable and robust.
5. Battery or mains powered.
6. Able to recognise a variety of syringe types and sizes.
7. Rapid infusion facility up to 1200 ml/hour.
8. Able to communicate with controllers (RS232 interface).
9. Able to alert user of line occlusion and need to recharge syringe.
10. Displays rate of infusion and volume infused clearly.

sion-engineered production model of the Ohmeda 9000 will possess this defect.

Evaluation of the Ohmeda 9000 pump during propofol anaesthesia was completed in 180 cases. Infusion induction with propofol at 5, 10 or 10 ml/minute was well tolerated by patients. The pump was used in the battery-operated mode during induction in the anaesthetic room, and maintenance infusions were continued during transfer into the operating theatre so that anaesthesia was not interrupted. The rapid infusion facility enabled propofol to be delivered quickly in the event of light anaesthesia.

Discussion

Syringe drivers have evolved as the most practicable and accurate devices for delivering drugs by infusion and are widely used in Intensive Care Units, but only recently has attention been focused on what the anaesthetist requires from an infusion device. The introduction of anaesthetic agents such as propofol and alfentanil have stimulated interest in infusional anaesthesia and the pharmacokinetics of intravenous drug infusions. The importance of the ability to establish rapidly a steady state concentration of drug without overshoot,²⁻⁴ and then vary it with ease, are now appreciated. A pump must be capable of infusion with accuracy at fast rates for short periods in order to fulfil these requirements. Computer-assisted infusion regimens capable of achieving pharmacokinetic objectives⁵⁻⁷ and maintaining closed-loop control of physiological variables⁸⁻⁹ are now available. An infusion device must be able to communicate with a controller in order to use these regimens.

The performance characteristics of the Ohmeda 9000 syringe pump can be summarised in relation to desirable properties of an infusion device for anaesthesia (Table 3). Mechanical failure did not occur during the study period and the pump was extremely reliable. The backbar contains a step-down transformer that provides electrical isolation. The Ohmeda 9000 pump complies with BS5724 safety requirements.

Over the range of infusion rates studied (2–90 ml/hour), delivery was accurate to within 5% of the predicted infusion volume compared with the manufacturer's claim of 3%. Greater inaccuracy was encountered using BD Plastipak 10-ml syringes, which consistently underdelivered at the lowest infusion rate. The occurrence of 'stiction' in the syringe appeared to be the major contributory factor, rather than inaccuracy in the pump transmission system.

Several features are incorporated in the Ohmeda 9000 designed to facilitate its use by the single-handed anaesthe-

tist. The touch key pads and rotary dial made set-up and operation fast and uncomplicated. The snap-fit syringe cradle allowed syringes to be inserted and removed with ease using one hand, but the retainer for the syringe plunger was less easy to manoeuvre. The carriage mechanism must be disengaged by opening the snap-fit retainer for the syringe plunger in order to reset it during syringe replacement. This proved to be excessively stiff in the prototype tested and required the use of a second hand to stabilise the pump. The tongue-and-groove connexion between the pump and backbar made insertion and removal of the pump easy, although care must be taken to ensure that both sides of the pump are correctly located. The clamp that secured the backbar to the drip-stand was poorly designed and occasionally worked loose during syringe replacement.

The Ohmeda 9000 is a compact and light-weight pump and can easily be carried in one hand. The transfer of patients with infusions in progress is a hazardous time for pumps if they are not secured to a drip-stand or other immovable object. The prototype Ohmeda 9000 functioned normally after one (uncontrolled!) fall and the production model will doubtless be more robust.

No alarms for low battery charge were encountered when using the pump in the battery-operated mode for periods of up to 4 hours. No recharging of the batteries was undertaken, other than when the pump was operating on mains power.

The pump successfully recognised the size of all syringes used during the study period thereby simplifying set-up procedures. The only exception was the Braun Omnifix 30-ml syringe that was accepted as a 20-ml syringe. This resulted in 30% inaccuracy of delivery because of excessive infusion. It should be emphasised that the Ohmeda 9000 is calibrated exclusively for Becton Dickinson, Sherwood and Terumo 10, 20 or 50/60 ml syringes and only syringes purchased from the recommended manufacturers should be used, or drug delivery will be subject to inaccuracies. It is unlikely that more than one make of syringe will be readily available in a fixed location. Terumo syringes were not tested in the present study.

The rapid infusion facility provided by the pump was easy to adjust ensuring prompt delivery during clinical anaesthesia. The real-time LCD of bolus volume delivered was particularly useful as was the audio tone which marked the delivery of each millilitre of infusate. Rapid priming of the infusion line could also be achieved with this facility.

The LCDs of infusion rate and volume infused were easy to visualise and were reliable. The two-level alarms for syringe near-empty and empty allowed syringes to be replaced with the minimum interruption of drug delivery in the clinical setting. The line pressure alarm gave prompt warning about occlusions in the delivery system, and was also activated during continuous infusion using 10-ml syringes that were impeded by 'stiction'. The occlusion alarm was not activated by the increase in line pressure resulting from bolus infusions because of simultaneous adjustment of the alarm threshold from 700 to 1200 mmHg. The prototype used in this study was not interfaceable so that evaluation of the computer-controlled infusion mode was not possible.

The Ohmeda 9000 syringe pump appears to fulfil all the requirements of an infusion device for anaesthesia, and clinical evaluation proved it to be easy to use and reliable.

It should receive wide acceptance by anaesthetists who practise infusion anaesthesia.

Acknowledgments

The authors thank Mr L. Smith of Ohmeda (BOC Health Care) for providing prototypes of the Ohmeda 9000 pump and Dr I. Glen of ICI Pharmaceuticals for his assistance.

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Use of the oesophageal detector device in children under one year of age

S. R. HAYNES AND N. S. MORTON

Summary

The efficacy of a modified oesophageal detector device was evaluated in a single-blind study of 20 healthy infants. It was found to be unreliable as a method to discriminate oesophageal from tracheal intubation in this age group.

Key words

*Equipment; oesophageal detector device.
Anaesthesia; paediatric.*

Tracheal intubations may be performed in an emergency in children under one year of age by clinicians who may not have received any formal anaesthetic training. A rapid, reliable method to discriminate between oesophageal and tracheal tube placement would be of value to these clinicians as well as to anaesthetists.

The oesophageal detector device was shown reliably to distinguish oesophageal from tracheal tube placement in adults,¹ and also in children aged 5–10 years intubated with uncuffed tubes.² It consists of a 60-ml syringe attached to a tracheal tube connector. Air can easily be aspirated when connected to a tube in the trachea, and when connected to a tube in the oesophagus, air cannot be aspirated.

The aim of this study is to assess a modified oesophageal detector device for use in children under one year of age.

Methods

Twenty patients aged under one year were studied. All were ASA grade 1 or 2 and were to receive anaesthesia for elective surgery. Approval for the study was granted by the local hospital ethics committee, and informed parental consent was obtained.

Anaesthesia was induced by either an intravenous or an inhalational technique as appropriate. Tracheal intubation was performed with the aid of a muscle relaxant, according to normal practice. Correct tracheal tube placement was checked by the routine method of direct vision, auscultation, and observation of the chest wall in response to

ventilation, and after satisfactory maintenance of anaesthesia was achieved, a second, identical tube, was passed into the oesophagus. The position of the tube on the left-hand side of the patient's mouth was randomised to be either oesophageal or tracheal. The anaesthetic breathing system was disconnected and a towel with a small window placed across the child's face so that only the left-hand tube connector was visible. An observer blinded to the procedure so far was then asked to aspirate from the tube connector presented through the towel window using the modified oesophageal detector device described below. The breathing system was reconnected and the anaesthetic and operation allowed to proceed as normal. The observer was asked to indicate before reconnection the apparent position of the tube presented to him. A pulse oximeter was used to monitor each patient during the disconnection, and on no occasion did the oxygen saturation decrease below 95%. All patients were seen after operation and no sequelae were identified.

Modification to the original oesophageal detector device

The original device was constructed using a 60-ml syringe. We believed that this was too big for this age group since there would be a risk of aspirating an excessive proportion of a small anaesthetised subject's functional residual capacity. Our modification (Fig. 1), consisted of a straight Portex 15-mm female connector, joined by a short length of corrugated antistatic tubing to a disposable 5-ml Plastipak syringe, so that the nozzle of the syringe was within the

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Accepted 4 April 1990.

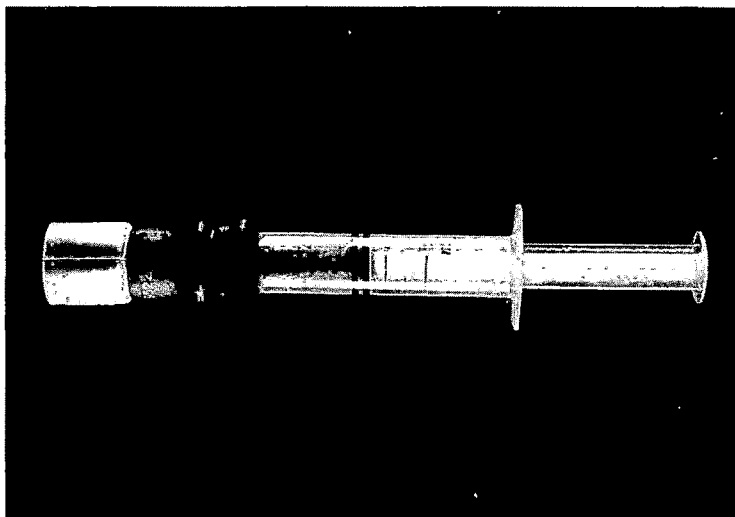


Fig. 1. Modified oesophageal detector device for use in children under one year of age.

proximal portion of the tube connector. The deadspace of the apparatus is 3 ml, which is reduced when a 15-mm male tracheal tube connector is joined, as when the device is in use.

Results

In eight infants aged 2 weeks to 9 months, who weighed 4.6 to 9.2 kg, tracheal tube placement was correctly identified,

but in two further infants tubes placed in the trachea were wrongly thought by the observers to be in the oesophagus (Table 1).

In seven infants aged 6 days to 10 months, who weighed 3.1 to 9.0 kg, oesophageal tube placement was correctly identified, but in three infants, tubes placed in the oesophagus were wrongly thought by the observers to be in the trachea (Table 2).

It should be stressed that the observers were blinded to

Table 1. Tracheal tube placement ($n = 10$). Mean age (SD), 4.8 months (2.7); mean weight (SD), 7.4 kg (1.8).

Patient number	Age	Weight (kg)	Tube size (mm)	Observer	Response
1	2 weeks	4.6	3.0	Registrar anaesthetist	Correct
2	4 months	8.8	4.0	Registrar anaesthetist	Correct
3	2 months	5.6	3.5	Senior Registrar anaesthetist	Wrong
4	3 months	7.7	3.5	Registrar anaesthetist	Correct
5	5 months	8.3	4.0	Registrar anaesthetist	Wrong
6	9 months	9.2	4.0	Registrar anaesthetist	Correct
7	4 months	6.8	4.5	Registrar anaesthetist	Correct
8	6 months	9.0	4.0	Registrar anaesthetist	Correct
9	7 months	9.1	4.0	Senior Registrar anaesthetist	Correct
10	4 weeks	4.7	3.5	Senior Registrar anaesthetist	Correct

Table 2. Oesophageal tube placement ($n = 10$). Mean age (SD), 3.95 months (3.40); mean weight (SD), 5.89 kg (1.97).

Patient number	Age	Weight (kg)	Tube size (mm)	Observer	Response
1	5 months	6.0	3.5	Surgical Registrar	Correct
2	5 months	7.6	4.0	Consultant anaesthetist	Correct
3	4 weeks	4.6	3.5	Registrar anaesthetist	Correct
4	9 months	9.0	4.0	Registrar anaesthetist	Wrong
5	4 months	4.6	3.5	Registrar anaesthetist	Correct
6	3 months	6.4	3.5	Registrar anaesthetist	Wrong
7	3 weeks	4.6	3.5	Senior Registrar anaesthetist	Correct
8	6 days	3.1	3.0	Consultant anaesthetist	Correct
9	6 weeks	4.0	3.5	Orthopaedic Registrar	Correct
10	10 months	9.0	3.5	Orthopaedic Registrar	Wrong

the tube position and used only the detector device to make their decision.

The observers who failed in two cases to identify correct tracheal placement of the tube were an anaesthetic registrar and an anaesthetic senior registrar. Both individuals had correctly identified oesophageal and tracheal tube placements on other patients in the study. The observers who failed to identify oesophageal tube placement were two anaesthetic registrars and an orthopaedic registrar, all three of whom had correctly identified tube placement, using the device, on other patients in the study.

The principle underlying the oesophageal detector device is that the trachea is held open by rigid cartilaginous rings, to allow free aspiration of the gas within the respiratory tract. The oesophagus is not supported and collapses when a negative pressure is applied within its lumen, thus preventing free aspiration of air.¹ This principle in adults and older children renders the oesophageal detector device seemingly infallible,^{1,2} but the results of our study show a 25% failure rate of the modified oesophageal detector device. This clearly demonstrates that the same technique cannot be applied in children under one year.

There could be several reasons for failure to identify oesophageal tube placement in this age group using the modified oesophageal detector device. It is well established that there is a high incidence of gastro-oesophageal reflux and hiatus hernia in this age group,³ and it is conceivable that a tube positioned in the oesophagus in the presence of gastro-oesophageal reflux or hiatus hernia would allow gas to be aspirated from the stomach. Any prior ventilation by mask may increase the volume of intragastric gas, thus enhancing the likelihood of this happening. Another possible explanation is that the oesophagus does not

collapse so readily to form an airtight seal around tracheal tube, and a tracheal tube may split open the upper oesophagus thus enabling air to be aspirated from the pharynx. The larynx is relatively high in the neck in this age group, and a tube identical in length to the tracheal tube, when placed in the oesophagus, may only be a short distance within the oesophageal lumen, thus increasing the possibility of this happening. Conversely, it is conceivable that in some cases the oesophageal tube may have been passed into the stomach. This could have been possible in the smaller children studied. Failure to detect oesophageal placement occurred in children aged 3, 9 and 10 months who weighed respectively 6.4, 9.0 and 9.0 kg, so we believe that in these cases this is not a likely explanation.

Failure to identify tracheal tube placement indicates either that the tracheal wall is not held rigidly open by cartilage as in older children or that the mucosal lining of the trachea can collapse over the tube when a negative intraluminal pressure is applied.

The results suggest that in its present form the oesophageal detector device cannot be recommended for use in children under one year of age.

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Nasal pulse oximetry overestimates oxygen saturation

J. ROSENBERG AND M. H. PEDERSEN

Summary

Ten surgical patients were monitored with nasal and finger pulse oximetry (Nellcor N-200) for five study periods with alternating mouth and nasal breathing and switching of cables and sensors. Nasal pulse oximetry was found to overestimate arterial oxygen saturation by 4.7 (SD 1.4%) (bias and precision).

Keywords

Equipment; pulse oximeter.
Oxygen; hypoxaemia.

Pulse oximetry is a commonly used technique for monitoring oxygenation in the operating theatre, recovery room and intensive care unit and was shown to give accurate estimation of arterial oxygen saturation over a wide range of values using both finger and ear probes.^{1,2} It was suggested that nasal pulse oximetry could be of value in patients with an impaired peripheral circulation.^{3,4} Nasal probes function with the same empirical calibration algorithm as other probes and are thought to give reliable oximetry readings, but our preliminary findings suggested that oxygen saturation may be overestimated by 5–10% with this method.⁵ We therefore conducted a trial on the accuracy of nasal pulse oximetry.

Methods

Ten patients, median age 78 (range 45–84 years), weight 68 (37–106 kg), height 170 (140–176 cm), were studied. The surgical department was screened to find patients with nasal pulse oximetry oxygen saturation (SpO_2) below 100%. Informed consent was obtained from the patients who entered the study, seven of whom were in the postoperative period (days 2–15). Two patients had gastric surgery, three colonic resection, one cholecystectomy and one appendectomy. Three patients had not undergone surgery. Post-operative analgesia comprised intramuscular morphine on demand. Median opioid administration in the preceding 8 hours before the study was 0 mg morphine (range 0–10). All patients who had undergone surgery had received general anaesthesia. They were not clinically dehydrated, did not receive oxygen, and were peripherally warm with no signs of cyanosis.

Two Nellcor N-200 pulse oximeters were used (software version 2.5) with a nasal (adult nasal oxygen transducer R-15) and a finger probe (adult digit oxygen transducer D-25). Oxygen saturations were simultaneously recorded on a penwriter.

The study consisted of five periods of 15 minutes each. The patient was monitored in period one, with the digit and nasal probes, breathing through the nose; in period two, the nose was plugged in order to abolish nasal airflow; in period three, cables between the probes and the amplifier box were switched; in period four, cables between the amplifier box and oximeters were switched; in period five, the digit and nasal probes were replaced with new probes. The probes were attached according to instructions,⁶ and C-lock was used in all patients in order to minimise motion artefacts.⁷ An arterial puncture was performed and the blood gases measured using an ABL300, Radiometer A/S, at the beginning and end of the study.

The study was in accordance with Helsinki Declaration II, and approved by the local ethics committee. Wilcoxon's, Mann-Whitney's and Spearman's tests were used for statistical analyses. Statistical significance was chosen at $p < 0.05$.

Results

There was no difference between nasal SpO_2 ($p > 0.16$) or finger SpO_2 ($p > 0.79$) in period one compared to period two, or between nasal SpO_2 ($p > 0.16$) or finger SpO_2 ($p > 0.12$) in period four compared to period five (Fig. 1.). The median values for nasal and finger pulse oximetry are shown in Table 1.

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Accepted 15 February 1990.

Table 1.

	Period 1	Period 2	Period 3	Period 4	Period 5
Nasal SpO ₂ ; % (range)	96.2 (89.4–97.4)	96.5 (90.8–98.3)	95.6 (90.4–97.5)	96.4 (91.5–99.0)	96.0 (91.0–98.7)
Finger SpO ₂ ; % (range)	92.2 (86.4–95.0)	91.8 (88.3–95.0)	92.3 (87.6–94.5)	92.5 (87.1–95.0)	91.8 (85.4–93.5)
	p < 0.003	p < 0.002	p < 0.006	p < 0.002	p < 0.004

Median overall difference between finger and nasal SpO₂ was 3.9% (range 0.1–7.0).

Finger SpO₂ correlated with arterial oxygen saturation ($r_s = 0.76$, $p < 0.03$), and the values did not differ significantly (Wilcoxon, $p > 0.5$). Nasal SpO₂ did not correlate with ($r_s = 0.64$, $p > 0.05$) and was significantly higher than the arterial oxygen saturation (Wilcoxon, $p < 0.006$). Thus, the bias and precision² was 1.0 (SD 0.7%) and 4.7 (SD 1.4%) for the finger and nasal probe, respectively. Nasal SpO₂ correlated with finger SpO₂ ($r_s = 0.80$, $p < 0.02$), but the values differed significantly (Wilcoxon, $p < 0.006$).

Discussion

Nasal pulse oximetry has been suggested to be of value in patients with vasoconstriction or hypotension, because the anterior ethmoid arterial supply to the nasal septum persists over a wider range of pressures than the finger pulse.³ Nevertheless, the only available study employing nasal pulse oximetry showed that the finger probe was in fact more accurate than the nasal probe under such circumstances.⁴

The reason for the observed overestimation at the nose is unknown. Ambient light may affect the measurements,⁸ but the heart rate on the oximeter was stable, we were careful about the sensor adhering snugly to the skin, and no light leaked from the light source or ambient light to the sensor, so we can reject this explanation.

Reflection might affect light absorption and cause the pulse oximeter to overestimate oxygen saturation since the emitting diode and the detector on the nose are not exactly opposite each other. Therefore, if the sensor is used on another part of the body⁹ special care must be taken in probe positioning. Development of a disposable probe for intranasal monitoring on the septum may be a solution to the problems of nasal pulse oximetry outlined in this study.

Our results show that the nasal estimations of arterial oxygen saturation are consistently too high, whereas the finger probe gives an acceptable estimation of arterial oxygen saturation, as shown in previous studies.²

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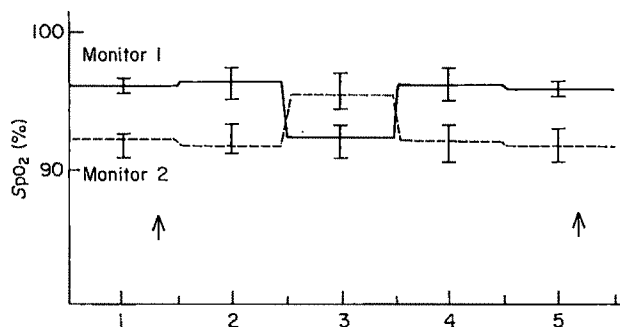


Fig. 1. Pulse oximetry oxygen saturation (SpO₂) for monitor one and two in the five study periods. The patient was breathing through the nose in period one; in period two, the nose was plugged in order to abolish nasal airflow; in period three, cables between the probes and the amplifier box were switched; in period four, cables between the amplifier box and oximeters were switched; in period five, the digit and nasal probes were replaced with new probes. Values are given as medians and quartiles. Arrows indicate arterial puncture. —, nasal probe; ---, digital probe.

Forum

Patients' expectations of patient-controlled analgesia

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Summary

Patient-controlled analgesia is an increasingly popular method of postoperative pain relief. However, patients often worry about new therapies. Eighty ASA 1 and 2 patients aged 18-65 years were asked to list the advantages and disadvantages of using patient-controlled analgesia. The most important advantage as perceived by patients was the reduced time spent by nurses in giving medication, but there was concern that direct personal contact would also be lessened. Preservation of self control, autonomy, rapid onset of analgesia, ability to titrate analgesia and lack of injections were seen as an advantage. Addiction and machine faults were seen as minimal problems. Preservation of patient-nurse contact is of great importance to ensure success of postoperative analgesia.

Key words:

*Pain; postoperative.
Analgesia; patient controlled.*

Patient-controlled analgesia (PCA) is an increasingly popular method of postoperative pain relief. Interest in this mode of opioid delivery stems from the well documented inability of medical and nursing staff to administer analgesics adequately,^{1,2} together with failure to achieve adequate pain relief once these drugs are delivered.^{2,3} Much has been written about assessment of patients postoperatively, although there is very little reported work on how patients perceive the importance of postoperative pain relief, their hopes of its efficacy and worries about its side effects. The importance of postoperative pain relief was highlighted in a recent report, 'Management of Severe Pain',⁴ by the Australian National Health and Medical Research Council.

Patients' expectations of postoperative pain relief can be formed by education before operation. However, general information sheets, for example the Australian Society of Anaesthetists' Information Sheet for patients undergoing anaesthesia, provides just four lines on postoperative management and analgesia and are of little value. Some companies who manufacture PCA hardware, produce information sheets or booklets that provide information for patients, but do these answer patients' anxieties?

The aim of this study was to examine the expectations of patients about patient-controlled analgesia, to document any misgivings they might have and to provide guidelines for better information to compliment PCA.

Methods

Eighty female patients, aged 18-65 years who were scheduled for elective gynaecological surgery were asked to take part in the survey after Ethics Committee approval.

Patients were given standardised descriptions of PCA and conventional intramuscular therapy during the pre-operative visit by their anaesthetist. No bias was suggested about either therapy. It was stressed that with both options, pain-relieving medication could be obtained at the push of a button; (either the PCA pendant or the nurse-call bell-push). They were then given a questionnaire that was to be completed later, but before they went to the operating theatre.

The questions asked were: 'We would like to have your opinions on the advantages/disadvantages of using the equipment which allows patients control of their own pain relief (PCA). In the spaces below (there were five for both advantages or disadvantages) list any advantage/disadvantage in using PCA, from your point of view.' Questions were designed to collect individual feelings rather than factual recall of pre-operative information by the anaesthetist. All views expressed were solely those of the patients.

The questionnaires were reviewed and major categories of response identified. The answers were again examined and responses put into the appropriate category.

Results

Completed questionnaires were collected from 74 patients (92.5%). Eighteen categories were derived for the 'Advantages' group (Table 1) and 13 for the 'Disadvantages' group (Table 2).

The benefit of PCA most frequently cited by patients was that it minimised the amount of time that nurses needed to spend administering analgesia (37.5%). The rapid onset of

Table 1. Advantages of PCA (n=74).

Reason	Number
1. Not bothering nurses, nurses too busy with others	30 (37.5%)
2. Rapid onset of pain relief	27 (33.8%)
3. In control of own pain relief	16 (20%)
4. Self-control, not losing autonomy	15 (18.8%)
5. Titrate exactly to needs	15 (18.8%)
6. Lack of injections	8 (10%)
7. No benefit	8 (10%)
8. Independence	5 (6.3%)
9. Reduction in the amount of pain	4 (5%)
10. Reassurance	4 (5%)
11. Not relying on nurses assessment of pain	4 (5%)
12. Stable pain relief	3 (3.8%)
13. Helps research	3 (3.8%)
14. Mobilisation better	2 (2.5%)
15. Will not worry other patients	2 (2.5%)
16. Reduction in amount of drugs	1 (1.3%)
17. Privacy	1 (1.3%)
18. Control of nausea	1 (1.3%)

analgesia (33.8%), ability to self-control pain relief (20%) and ease of titrating pain relief (18.8%) were also of major importance. The ability of the patient to retain autonomy (18.8%), independence (6.3%) and reassurance (5%) were other factors of note. One in 20 patients believed that it was important for themselves to gauge pain, rather than convince the nursing staff of their need for analgesia. Reduction in the number of injections was also seen to be an advantage (10%).

Under half the patients (45%) considered there would be no major disadvantage when using PCA. The well recognised worries about overdose (11.3%), addiction (3.8%) and machine dysfunction (6.3%) were expressed by a number of patients. Others were worried that the use of PCA would restrict their access to nursing care (11.3%). The potential to over-use PCA, and give themselves 'too much pain relief' was expressed by 10% of patients, while the possibilities of over-sedation (2.5%), expense (1.3%), restriction of movement (1.3%) and insecurity (2.5%) were less common worries.

Discussion

The main interest was the attitude of patients to post-operative analgesia. The reduction, with PCA, in the amount of time nurses needed to spend in administering postoperative analgesic agents was important, i.e. it appears that pain relief is perceived as less important than other activities of the nurse on the ward. 'I don't want to bother the nurse; she will be too busy attending to patients with greater problems than myself' were common replies. However, one of the main disadvantages of PCA was the worry that the patient/nurse contact would be reduced. This, in some ways, was verified in a number of studies where it was shown that time spent in analgesic-related activities is reduced when PCA is compared to conventional medication; Levi² reported that for a 4-day stay in hospital, there was a 20% reduction in time spent on administration of analgesics in the PCA group compared to the conventional parenteral therapy group. Theoretically, this should lead to increased nursing time in other facets of the nursing care plan, although studies are lacking to show this is indeed the case.

The control and individual titration of pain relief was seen as an advantage, although a number of patients expressed a worry that they would give themselves too much painkiller: 'I could give into the pain more; I may

Table 2. Disadvantages of PCA.

Reason	Number
1. No disadvantage	36 (45%)
2. Overdose	9 (11.3%)
3. Lack of nurse contact, less personal contact	9 (11.3%)
4. Over use, taking too much	8 (10%)
5. Machine dysfunction	5 (6.3%)
6. Inadequate analgesia	5 (6.3%)
7. Addiction	3 (3.8%)
8. Insecurity	2 (2.5%)
9. Over sedation	2 (2.5%)
10. No compassion	2 (2.5%)
11. Expense	1 (1.3%)
12. Restrict movement	1 (1.3%)
13. Needs intravenous cannula	1 (1.3%)

need it longer than allowed'. The fear of losing control is worse for those who feel a need to maintain a stoical image in the face of pain or adversity.⁶ Control is not beneficial for all patients; some would prefer decision-making to remain in the hands of the nursing staff.

Only 5% of patients perceived that PCA would give them a reduction in the total amount of pain during the peri-operative period. It seems that factors other than analgesia play an important role in their assessment of success of postoperative management e.g. independence, reassurance, self-control and privacy. Injections and the fear associated with intramuscular therapy are a major worry of the hospitalised child.⁷ This fear is also present in the adult population and PCA is useful in reducing this anxiety. A large proportion of the patients studied could think of no disadvantage from PCA therapy, with the fear of addiction, machine dysfunction and over-sedation rarely mentioned.

These results indicate how we can better explain post-operative pain therapies and relieve the apprehensions of the patients before operation. PCA information sheets and booklets currently comment mostly on the problems of technical errors, overdose and addiction potential. They reflect medical attitudes and appear to be written without the benefit of a systematic study of patients' views. It needs to be emphasised that the patients' individuality will be maintained, that time spent with direct nurse contact will not be reduced, and that the machine will not, and never was intended to, replace the need for continuing personal assessment by nursing staff. The marketing of PCA as a method of saving nursing time is decried, unless this 'saved' time is spent with patients; it should never be seen as a way to reduce staffing levels further.

Increasing use of technology in the quest for optimum therapy is common in modern medicine. It is clear that traditional basic nursing care and personal contact are still of paramount importance to patients. This should not be forgotten with PCA.

Acknowledgments

This study was supported in part by the CIG/Australian Society of Anaesthetists' equipment award. The cooperation of the anaesthetists and gynaecologists at Flinders Medical Centre was appreciated.

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Anaesthesia, 1990, Volume 46, pages 1074–1076

Patient-controlled analgesia in children

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Summary

We report our experience in introducing patient-controlled analgesia at the Royal Hospital for Sick Children, Glasgow. Twenty-five children used the technique after orthopaedic or general surgery using the Graseby system. The pump was loaded with 1 mg/kg morphine sulphate in 50 ml. A bolus dose of 0.02 mg/kg (1 ml) and a lockout interval of 10 minutes were the initial settings. The dose used, pain and sedation scores, respiratory rate and arterial oxygen saturation were recorded. Ages ranged from 5–15 years (mean 9.6) and the method was used for a mean of 48 hours after operation. Morphine requirements averaged 26 ($\mu\text{g/kg}$)/hour (SD 10.6). Pain control was good and sedation minimal. Adverse effects were few and minor. Education of patients, parents and nurses is essential for its success and safety. The technique is an effective and safe means of providing good quality analgesia in school age children.

Key words

Pain; postoperative.
Anaesthesia; paediatric.

Patient-controlled analgesia (PCA) is a safe and effective method of providing postoperative pain relief.¹ It has been extensively used in studies of adult patients and was shown to be effective in adolescents and, more recently, in children aged 5 years and over.^{2–6} We have recently introduced this technique at our hospital for postoperative paediatric patients and wish to report the findings in our first 25 children. Like Gaukroger *et al.*,⁴ we allowed any child able to understand the idea and physically to operate the equipment to use PCA for as long as opioid analgesia was required in the postoperative period.

Methods

The study was approved by the hospital ethics committee. The Graseby PCAS machine (Graseby Medical Ltd.) was used. The dose was standardised by diluting 1 mg morphine/kg lean body weight with 50 ml 0.45% saline, giving a concentration of 0.02 mg/ml. A bolus dose of 0.02 mg/kg was used and the lockout interval set at 10 minutes. These values were adjusted if required. Only the anaesthetist changed the settings or the syringes. No background infusion was used. A one-way valve in the intravenous infusion line ensured that morphine was delivered to the patient and not to the infusion bag.

Children were considered suitable if they were to have

orthopaedic or general surgery likely to require repeated doses of opioid analgesics and if they were able to understand the concept of PCA. Only children able to operate the trigger of the PCA device easily were studied. Patients and their parents were taught about PCA the day before surgery when possible, but in a few cases this teaching was given postoperatively but before the start of PCA. Patients were instructed to use the trigger as often as they liked to keep themselves comfortable but also to allow a few minutes between tries to assess the effect of each dose. It was emphasised to parents and nurses that only the child should operate the pump. Nurses who cared for the patients were individually taught at the time PCA was started.

There was no attempt to standardise anaesthetic techniques and in particular regional blocks were used where appropriate. Patients were made comfortable in the recovery room with titrated doses of intravenous morphine if required then PCA was started when the patient returned to the ward. Nurses recorded respiratory rate, oxygen saturation, pain and sedation scores hourly in addition to standard postoperative monitoring. The number of triggering attempts, the percentage of these which were successful and the volume left in the syringe were recorded for the last 19 patients. A three-point verbal reporting pain score (1, not really sore; 2, quite sore; 3, very sore) and a

Table 1.

Type of operation	n
Osteotomy	6
Extension of Tendo Achilles	1
Scoliosis surgery	1
Appendicectomy	4
Renal and ureteric surgery	6
Orchidopexy	3
Laparotomy	2
Thoracotomy	1
Burns (no surgery)	1
Total	25

four-point sedation score (1, eyes open spontaneously; 2, eyes open to command; 3, eyes open to shaking; 4, unrousable) were used. Patients were not deliberately roused unless the nurse suspected oversedation. A recording of zero denoted that no scoring had been attempted. A named anaesthetist was available at all times and visited the patient regularly to check that the technique was working satisfactorily. PCA was continued until it was considered oral analgesics would be adequate. The nursing staff were instructed to call the anaesthetist if the patient was unrousable, in severe pain or excessively nauseated; if the pump was alarming or not operating; if the intravenous line was not functioning or if the arterial oxygen saturation was persistently low.

Results

Fourteen male and 11 female patients aged 5–15 years (mean 9.6, SD 2.8) were studied. The mean weight was 30.7 kg (SD 10.4, range 15–60 kg). PCA lasted from 17 to 110 hours (mean 48 hours, SD 24), starting when the first dose was taken. The procedures performed are shown in Table 1.

The mean hourly morphine consumption (SD) was 32.5 (14.6) $\mu\text{g/kg}$ for the first 6 hours, 30.1 (14.9) $\mu\text{g/kg}$ for the first 24 hours and 26 (10.6) $\mu\text{g/kg}$ for the total PCA period (Table 2). The mean pain score (SD) was 1.7 (0.5) and the mean sedation score 1.5 (0.4) for the first 6 hours, decreasing to 1.5 (0.3) and 1.2 (0.3) respectively for the total duration of PCA (Table 2). Ten patients had at least one pain score of 3 (mean 3 scores each), and scores of 3 totalled 4% of pain observations. No patient had a sedation score of 4. These figures are based on observations made for 60% of the study hours, that is patients were asleep during 40% of the hourly observations when a recording of zero was made.

One patient had a minimum respiratory rate of 10 breaths/minute on one occasion only and this was not associated with either excessive sedation or oxygen desaturation. Otherwise respiratory rates ranged from a mean (SD) minimum of 17.6 (2.4) to a mean maximum of 30 (6.6). Oxygen desaturation ($\text{SpO}_2 < 90\%$) occurred in four patients. Two of these were single recordings of 89%. The

Table 2. Mean hourly morphine consumption, sedation and pain scores ($n=25$).

	Morphine dose ($\mu\text{g/kg/hour}$) mean (SD)	Sedation score mean (SD)	Pain score mean (SD)
First 6 hours	32.5 (14.6)	1.5 (0.4)	1.7 (0.5)
First 24 hours	30.1 (14.9)	1.2 (0.3)	1.5 (0.3)
Overall	26.0 (10.6)	1.2 (0.1)	1.4 (0.3)

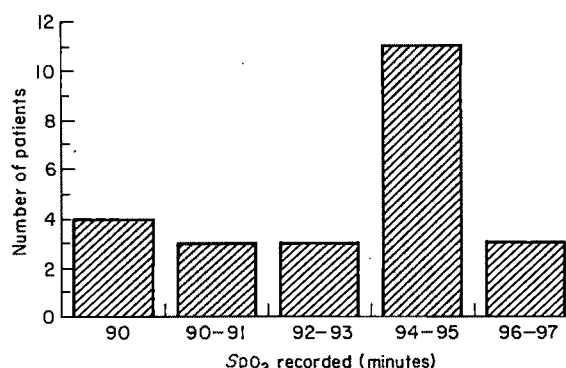


Fig. 1. Distribution of minimum oxygen saturation recordings.

other two were clearly related to surgical complications. One was a 12-year-old girl who developed ileus and abdominal distension 30 hours after surgery for an appendix abscess (SpO_2 84–90 for 12 hours); the other was a 12-year-old girl who had a small pneumothorax created during her nephrectomy procedure whose SpO_2 was 85–90 for two periods of 4 hours. In neither case was the low SpO_2 associated with excessive morphine consumption or oversedation. Saturation data are not available for one patient. Minimum arterial oxygen saturations are shown in Figure 1.

The percentage of successful trigger presses varied from 23–94 and tended to improve with time in those with a low value. Only one patient had nausea and vomiting recorded as a side effect and no other adverse effects were found. PCA settings rarely required alteration.

Two patients, one aged 4 years who had never been in hospital before and one aged 6 years with numerous admissions, refused to cooperate with the pre-operative training or to use the pump after operation. These have not been included in the 25 above.

Generally patients, parent and nurses welcomed PCA, often volunteering enthusiastic support for the technique.

Discussion

The management of postoperative pain in children is difficult and too often not done well. This is partly the result of a reluctance to provide children with potent opioid analgesics, either for fear of inducing respiratory depression or in the mistaken belief that children do not feel as much pain, and partly because some children will not ask for analgesia if they suspect that it means another injection.^{7–10} A continuous intravenous infusion of opioid can provide a predictable blood level of analgesic and removes the need for further injections.^{11,12} However, analgesic requirements vary widely between individuals so we do not know what blood level to aim for. Adjustment of the rate of an infusion can reach a new constant blood level only slowly, so patient-controlled analgesia overcomes these problems of interindividual variability and represents a significant advance in paediatric pain control.

We found that children had widely varying dose requirements even in apparently similar cases e.g. two cases of appendicectomy aged 10 and 12 years had a fourfold difference in requirement. We found a mean morphine uptake of about 30 ($\mu\text{g/kg}$)/hour, which is slightly less than that found by Gaukroger *et al.*⁴ This may be because we did not use a background infusion, which has been found in adults to increase consumption without improving pain relief.^{13,14} The dose of (30 $\mu\text{g/kg}$)/hour is less than might normally be prescribed for intermittent intramuscular injection, and represents a dose of 3.6 mg for a 30-kg child in 4

hours. It is possible that a sense of control over their pain reduces dose requirements. Analgesia was adequate as indicated by the generally low pain scores recorded by our patients. Simple to use verbal reporting pain scores are a good method of assessing pain in children.¹⁵

We believe that PCA is a successful innovation at our hospital, but its introduction has highlighted a number of points. Use of the technique must be discussed with the surgeon, the patients and their parents. The anaesthetists at this stage retain control of PCA and carry out any necessary adjustments, but it is nurses who directly supervise PCA. It is important that every nurse (and ward doctor) must fully understand the technique. We have taught them personally and individually up till now, but it may be that teaching can be devolved to ward sisters or nursing tutors and that ward staff will assume control of PCA as the technique becomes established. It is also important that nurses continue to teach the children to use the PCA correctly after it is started, for maximum success. One 13-year-old boy mistook the PCA trigger for a nurse call button, but after a reminder he had no further difficulty. Nurses should be alert to this type of problem particularly when a patient is just started on PCA.

The level of monitoring required has yet to be established. The only serious risk is of respiratory depression secondary to an overdose. This might theoretically occur if the pump had been primed with the wrong dose, a child were exceptionally sensitive to opioids i.e. the pump settings were inappropriate for that case, or if someone other than the patient repeatedly activated the trigger. Mechanical or electrical failures remain a possibility. Monitoring should aim to detect this as well as other problems such as inadequate analgesia or nausea and vomiting. Most workers have used respiratory rate as a measure of respiratory depression, but this is known to be insensitive. Wheatley has recommended oxygen saturation when breathing air as a much more sensitive method of detecting postoperative respiratory depression.¹⁶

The incidence of postoperative hypoxaemia found during this study was interesting. Two of our patients had clinically significant hypoxaemia which we attribute to surgical complications as detailed above. However, lesser degrees of arterial oxygen desaturation were more common. We used only hourly recordings of arterial oxygen saturation, although minimum readings of less than 94% occurred in eight other patients: six had had laparotomies and in these children there seemed to be no relation to sleep; one was a child with facial burns and one had a leg-lengthening procedure; in these children desaturation did seem to occur during sleep. It is not clear whether desaturation is the result of intrapulmonary shunting secondary to anaesthesia and surgery or to episodes of obstructive sleep apnoea exacerbated by opioid analgesia. No patient had a clinically apparent problem other than the two children with surgical complications. Continuous oxygen saturation recording would have provided better information and further studies combining this with nasopharyngeal carbon dioxide and impedance plethysmography are required, particularly when comparisons are to be made with conventional analgesic regimes.

In conclusion we found PCA to be a safe and effective means of providing analgesia in most children down to 5 years of age provided that adequate training is given to patients and staff and that patients are monitored appropriately. We recommend that this should include regular, preferably continuous, assessment of the level of analgesia, sedation and arterial oxygen saturation, at least until the problem of oxygen desaturation can be clarified.

Acknowledgments

We thank Graseby Medical Ltd and Dr G.N.C. Kenny of the University Department of Anaesthesia at Glasgow Royal Infirmary for the provision of the PCA pumps used in the study.

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Paraesthesia with lumbar epidural catheters
A comparison of air and saline in a loss-of-resistance technique

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Summary

The epidural space was located in 32 obstetric patients using loss of resistance to air, while in a further 35 saline was used. The incidence of paraesthesia was 56% in the air group and 57% in the saline group. There was no significant difference between the groups in terms of other complications or in the quality of analgesia provided.

Key words:

*Anaesthetic techniques, regional; epidural.
Complications; paraesthesia.*

The use of lumbar epidurals in obstetric practice is widespread and the procedure for insertion of the epidural catheter is relatively well tolerated and not especially painful. One part of the technique which can cause discomfort is the passage of the catheter through the epidural needle, which frequently results in paraesthesia. Relatively few authors mention this phenomenon, but the published incidence ranges from most,¹ to 40 or 50%.²⁻⁵

The incidence of paraesthesia was shown to be reduced when the paramedian approach is used, both in obstetric² and nonobstetric populations.³ However the midline approach is still the most popular in this country.

Another study showed a reduction in the incidence of paraesthesia when 10 ml air was injected into the epidural space before catheter insertion.⁴ It is also believed that the use of saline may 'open up' the epidural space and make insertion of a catheter easier in difficult cases.⁶ There does not seem to be any formal appraisal of these two techniques, so we decided to compare the use of saline with air on the incidence of paraesthesia during passage of epidural catheters in an obstetric population.

Methods

The study was approved by the Hospital Ethics Committee. All women who required the insertion of a lumbar epidural catheter for the relief of pain in labour, Caesarean section or other obstetric procedure were eligible for inclusion in the study. The procedure was explained and informed consent obtained. The epidurals were performed by one of us (I.S.), with the patient in the sitting position; the operator was experienced at locating the epidural space with both air and saline. The nearest easily identifiable space to L_{2/3} was chosen.

The epidurals were performed with a 16-gauge disposable Tuohy needle and loss-of-resistance syringe (Portex Mini Pack No. 1). The syringe was filled with 10 ml of either air or sterile normal saline as determined by previous random selection. The operator could obviously not be blinded as to this choice, but neither the patient nor her accompanying midwife knew which was in use.

The bevel of the Tuohy needle was turned to face cephalad, after location of the epidural space, and the entire remaining contents of the syringe were injected. The catheter was then passed to 15 cm from the needle hub, between contractions, during which the patient was questioned as to the occurrence of paraesthesia, which was defined as an unpleasant tingling sensation in the back, or radiating into the legs. Paraesthesia, if it occurred, was graded as moderate or severe. The midwife, who was blind to the technique, ensured that the anaesthetist did not influence the patient's assessment.

The catheter was withdrawn, after removal of the needle, so as to leave 3 cm within the epidural space. All subsequent management followed the usual departmental policy. The occurrence of dural and vascular punctures as well as any corrective action taken was noted. The quality and height of the block was assessed after 20 minutes and the mother's overall assessment of the effectiveness of the epidural throughout the first and second stages of labour (where applicable) was obtained, 24 hours after delivery.

Results

A total of 67 patients were studied; 32 were in the air group. The two groups were comparable with regard to age, height, weight at booking and before delivery, and for the depth of the epidural space from the skin (Table 1).

Eighteen patients (56%) in the air group complained of paraesthesia, two of whom found it severe. Paraesthesia was experienced by 20 patients (57%) in the saline group; it was severe in four (Table 2). The difference was not significant.

Complications (Table 3). No dural puncture occurred in either group. There were three vascular punctures in the air group (9%) and two in the saline group (6%). All punctures occurred on insertion of the catheter and all were corrected by partial withdrawal and flushing with saline. No catheter needed resiting in either group. There was no consistent relationship between the occurrence of a vascular puncture and paraesthesia.

Difficulty in passing the epidural catheter was noted in

Table 1. Comparison of groups. Values are expressed as mean (SD).

	Air	Saline	Significance
Number	32	35	
Age, years	25.2 (4.7)	23.4 (5.0)	ns
Height, cm	162.2 (6.4)	161.0 (5.7)	ns
Booking weight, kg	64.3 (12.1)	66.5 (11.2)	ns
Late weight, kg	74.3 (12.1)	77.1 (11.6)	ns
Depth of space, cm	3.9 (0.8)	3.9 (0.7)	ns

Table 2. Incidence of paraesthesia on insertion of catheter.

	Air	Saline
No paraesthesia	14 (44%)	15 (43%)
Moderate paraesthesia	16 (50%)	16 (46%)
Severe paraesthesia	2 (6%)	4 (11%)
Total	32	35
Chi-squared	0.574	Degrees of freedom 2
p > 0.5		

Table 3. Incidence of complications.

	Air	Saline
Dural punctures	0	0
Vascular punctures	3 (9%)	2 (6%)
Chi-squared	0.324	p > 0.5
Catheter difficult to thread	4 (12.5%)	5 (14%)
Chi-squared	0.028	p > 0.5
Unblocked segment	1 (3%)	2 (5.7%)
Chi-squared	0.262	p > 0.5

five patients (14%) in the saline group and four (12.5%) in the air group. This was always associated with paraesthesia in the saline group (in one of whom there was also a vascular puncture), but two of the air group had no paraesthesia.

There was no difference between the groups with regard to quality of pain relief. An unblocked segment occurred in one patient in whom the space was located with air and two when saline had been used.

Discussion

The incidence of paraesthesia in this study is in keeping with the findings of other workers. We have failed to confirm the work of Philip⁴ that injection of air reduced the incidence of paraesthesia; our incidence of 56% with air was higher than his control value of 49%. Furthermore, we have shown that there is no difference between a compressible and a noncompressible fluid in their effect on paraesthesia, since the injection of 10 ml of air or saline into the epidural space, as part of a loss-of-resistance technique, produced equal incidences of paraesthesia.

Our sample size was not particularly large, but had either technique produced a clinically worthwhile reduction in paraesthesia, then a statistically significant result would have occurred, since the incidence of this complication is so high. The same is not necessarily true of phenomena with a lower incidence and therefore our results with regard to the occurrence of vascular punctures and the ease of catheter insertion should be interpreted with some caution.

However, there is no evidence of any dramatic differences between techniques.

The injection of 10 ml of bupivacaine before catheter insertion has previously been reported to reduce the incidence of vessel puncture from 9% to 3%,⁷ but there was no difference in the present study between air and saline. We have not found anything to support the notion that saline helps to 'open up' the epidural space.

There was generally a high level of satisfaction with the degree of analgesia provided. It was suggested that air in the epidural space may prevent uniform spread of local anaesthetic and lead to a patchy block.⁸ We noted only three unblocked segments in our study, which gave an overall incidence of 4.5%, smaller than the previously reported incidence.⁹ Two of these occurred in the saline group. Venous air embolism was also detected after the use of epidural air,¹⁰ but this does not appear to be of clinical significance. On the other hand, it was suggested that saline may dilute the local anaesthetic,⁴ reducing its effectiveness. We found no adverse effect of either technique upon outcome, and the choice between air or saline appears to remain a personal one.

Paraesthesia can be reduced by using the paramedian approach^{2,3} presumably because of the straighter and more midline course taken by the catheter when that approach is used.¹¹ A soft polyurethane catheter inserted via an unspecified route also reduced the incidence of paraesthesia from 44 to 24%.⁵ Hyperventilation by the subject during epidural catheter insertion was shown to have no effect on either the incidence of paraesthesia or vascular puncture.¹² The present study has shown that neither air nor saline injected via a midline approach has any effect on the occurrence of paraesthesia.

Acknowledgments

We thank Dr M.C. Clapham, Consultant Anaesthetist at East Birmingham Hospital for his help and encouragement during the planning and completion of this project. We also thank all of the midwifery staff at Marston Green Maternity Hospital for their help with patient assessment.

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Anaesthesia, 1990, Volume 45, pages 1079–1082

Effects of hypnotics on sleep and psychomotor performance A double-blind randomised study of lormetazepam, midazolam and zopiclone

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Summary

Lormetazepam, midazolam and zopiclone were compared as night medication in patients scheduled for elective surgery the next morning. Sixty patients divided at random into three groups, received double-blind lormetazepam 1 mg, midazolam 15 mg or zopiclone 7.5 mg, by mouth at 2200 hours. The quality of sleep was assessed at 0700 hours from responses to a questionnaire, and psychomotor function by comparing paper and pencil (p-deletion) and Maddox Wing tests with reference values from the day before. The three hypnotics were equally effective as sleep medication for time until onset of sleep, duration of sleep and condition upon awakening, whereas zopiclone provided significantly fewer ($p < 0.05$) spontaneous awakenings. The p-deletion test did not differ in any of the three groups from the reference values. The ocular imbalance test in all three groups was significantly different ($p < 0.01$) from control. The lormetazepam group scored significantly better ($p < 0.05$) than the zopiclone group. No side effects were seen.

Key words:

*Hypnotics; benzodiazepines.
Recovery; tests.*

The majority of hypnotics, especially benzodiazepines available as sedatives, anxiolytics or hypnotics are effective clinically as sleep-inducers. The ideal hypnotic drug should induce sleep without side effects and the patient should wake up without a hangover or disturbance in psychomotor performance. Benzodiazepines can, however, have insidious effects on human behaviour, leading to impairment of mental, cognitive and psychomotor performance in daily life.

Zopiclone, a new class of psychotherapeutic drug, is a cyclopyrrolone compound, which is chemically unrelated to benzodiazepines, but with binding sites on, or closely linked with the benzodiazepine receptor complex. It is metabolised to less active N-oxide derivate and inactive N-desmethyl zopiclone with a half-life of zopiclone, N-oxide metabolite and N-desmethyl zopiclone, respectively 3.5–6 and 7–11 hours.¹

Lormetazepam, a 3-hydroxybenzodiazepine, is a hypnotic drug with a half-life of 9.9 (SD, 2.4) hours. It is metabolised in the liver by conjugation and no active metabolites are formed, except lorazepam to a very minor extent.² Midazolam, also a benzodiazepine, with a half-life time of 2–3 hours³ is characterised by rapid clinical onset and short duration of action. It is metabolised by conjugation to hydroxy-midazolam which is pharmacologically active in animals, but without any significant clinical action.

The purpose of this study was to assess the quality of sleep and psychomotor performance after a single dose of

lormetazepam 1 mg, midazolam 15 mg or zopiclone 7.5 mg respectively.

Patients and methods

Sixty patients, 30 males and 30 females, in ASA groups 1 or 2, aged 18–65 years, scheduled for minor elective orthopaedic surgery of the upper and lower extremities participated in the investigation. The study was approved by the Medical Ethics Committee of the hospital, and all patients gave verbal informed consent. Recovery tests were performed to establish control values after assessment of the patients.

The patients were allocated at random into three groups. Group A ($n = 20$) received lormetazepam 1 mg; group B ($n = 20$) midazolam 15 mg and group C ($n = 20$) zopiclone 7.5 mg, by mouth as a capsule. Excluding factors were pre-operative pain, old age, pregnancy, allergic reactions to benzodiazepines, alcohol abuse, myasthenia gravis or other muscular diseases, and patients already using benzodiazepines or other hypnotic, antipsychotic medication. Administration of the hypnotic medication was at random and double blind. The patients received their medication the night before the operation at 2200 hours. The quality of sleep and psychomotor performance was assessed by the same observer, using methods similar to a previous study.⁴

Assessment of the quality of sleep. Patients were asked to complete a sleep-quality questionnaire, according to Wickström,⁵ that contained the following questions: length

Table 1. Patient age, sex ratio and weight; mean (SEM).

	Group A (lormetazepam) <i>n</i> = 20	Group B (midazolam) <i>n</i> = 20	Group C (zopiclone) <i>n</i> = 20
Females/males	11/9	11/9	8/12
Age; years	34.4 (2.5)	34.1 (2.6)	35.9 (3.0)
Weight; kg	68.1 (2.7)	69.5 (2.4)	73.9 (2.9)

of time before onset of sleep; duration of sleep; number of spontaneous awakenings; difficulty in falling asleep after awakening; patient's opinion of effectiveness of sleep medication; patient's evaluation of quality of sleep.

Assessment of psychomotor function. Measurement of ocular imbalance was performed as assessed by Maddox Wing;⁶ the p-deletion (pencil and paper) test was as described by Dixon and Thornton.⁷ This test was modified: each patient received a text that contained 140 letters p, and was asked to delete as many letters p as possible without omissions within 3 minutes. The total number of ps deleted was counted. The tests were performed the night before the hypnotic medication and the morning after at 0700 hours before premedication was given.

Statistical analysis was performed using analysis of vari-

ance for age, weight, sex, and p deletion. A Tukey test was used to compare the difference of the Maddox Wing results between the groups separately. Data concerning patients' opinions were analysed using the Kruskal-Wallis test. ($p < 0.05$ was considered to be significant.)

Results

The patients were comparable in age, sex and weight (Table 1). None of the patients received other medication likely to affect either psychomotor performance or hypnotic metabolism, for example H_2 receptor antagonists. The results of the subjective evaluation of the patients are listed in Figures 1-7.

A difference was found in the number of spontaneous

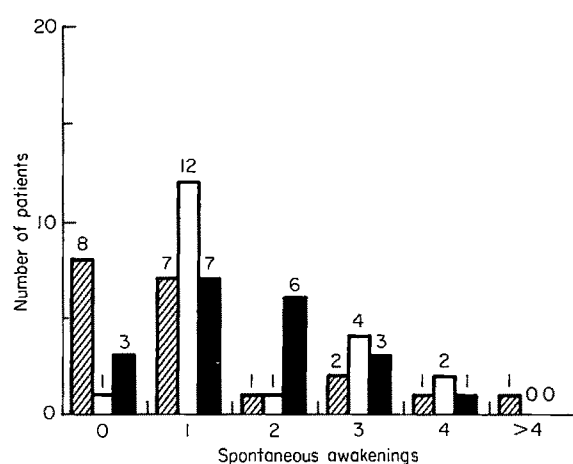


Fig. 1. The number of spontaneous awakenings. The zopiclone group showed significantly fewer spontaneous awakenings ($p < 0.05$) compared to the lormetazepam and midazolam groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

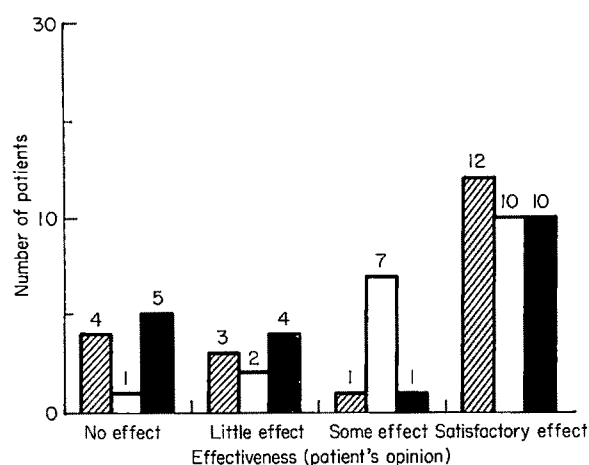


Fig. 2. Effectiveness of sleep medication. There were no significant differences between the groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

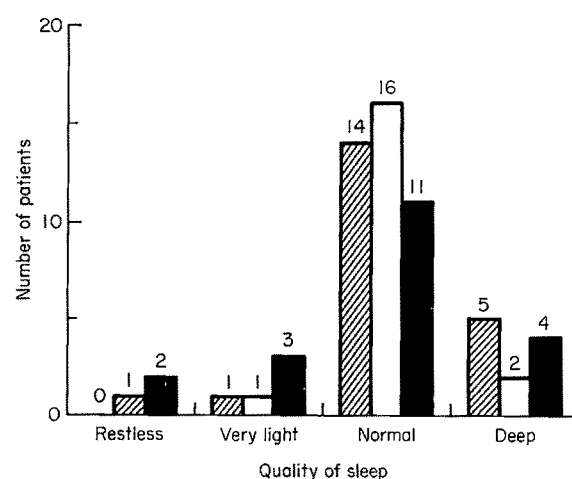


Fig. 3. Patients' opinion on quality of sleep. There were no significant differences between the groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

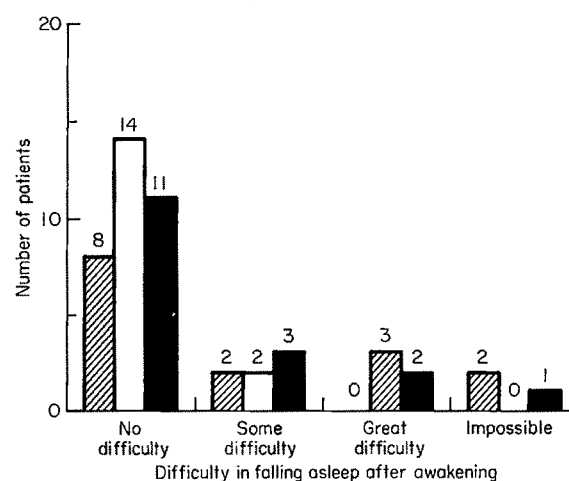


Fig. 4. Patients who had difficulty in falling asleep after awakening. There were no significant differences between the groups. ▨, zopiclone ($n = 12$); □, lormetazepam ($n = 19$); ■, midazolam ($n = 17$).

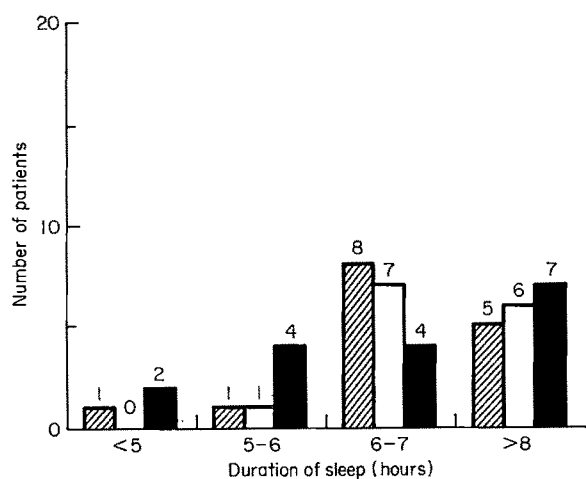


Fig. 5. Duration of sleep. There were no significant differences between the groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

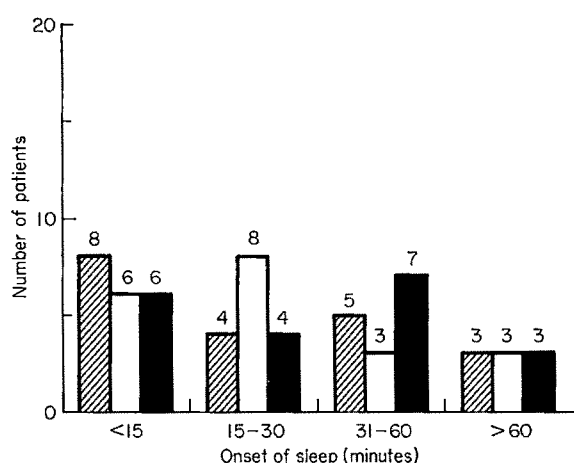


Fig. 6. Length of time before onset of sleep. There were no significant differences between the groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

awakenings (Fig. 1), of which there were significantly fewer in group C (zopiclone) compared to group A (lormetazepam) and group B (midazolam) ($p < 0.05$). No significant difference in length of time to onset of sleep, quality, duration or effectiveness of sleep, was found. The p-deletion score showed no significant difference between the three groups, or from control (Table 2).

The ocular imbalance test (Maddox Wing) was significantly different ($p < 0.01$) in all three groups compared to the reference values. However, the ocular imbalance test showed a significant ($p < 0.05$) difference between group A (lormetazepam) and group C (zopiclone); it was more disturbed in group C (Table 3).

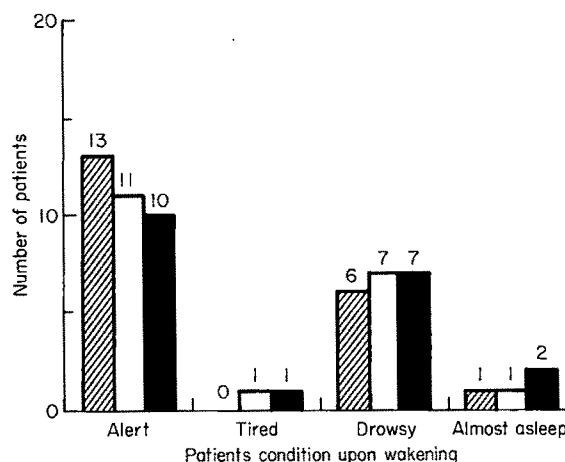


Fig. 7. Patients condition upon awakening. There were no significant differences between the groups. ▨, zopiclone; □, lormetazepam; ■, midazolam.

Eighteen patients in group A, 18 in group B and 20 in group C were awake at 0700 in the morning after the administration. No side effects occurred.

Discussion

The aim of the study was to assess quality of sleep and psychomotor performance (street fitness) the morning after the oral administration of lormetazepam 1 mg, midazolam 15 mg and zopiclone 7.5 mg the previous evening. The special circumstances of the night before the operation in relation to insomnia were thought to be comparable in patients scheduled for elective orthopaedic surgery. Two commonly prescribed benzodiazepines and a new psychotherapeutic drug, chemically unrelated to the benzodiazepines but with the same effect and receptor were chosen. Hypnotics without residual effects on psychomotor performance are to be preferred, either given as a single or repeated dose. A supportive visit by the anaesthetist and the use of sleep medication, benzodiazepines or other hypnotics, are frequently used in an attempt to reduce insomnia the night before operation.

Anaesthetists should be aware of the psychomotor effects of hypnotic drugs. Studies concerning hypnotics and performance have shown a relationship between traffic accidents and impaired psychomotor performance.⁸ To evaluate psychomotor performance (street fitness), two main basic functions can be distinguished: motor ability, and cognitive function. Test procedures to measure psychomotor performance are numerous, may vary widely, and no general guidelines are available. The ocular imbalance test by Maddox Wing as described by Korttila,⁹ and the p-deletion (paper and pencil test) test these basic functions.¹⁰

Table 2. Number of deleted ps; mean (SEM).

	Group A	Group B	Group C	Intergroup difference
<i>Control values</i>				
Deleted letters p	56.0 (3.5)	59.6 (4.2)	60.4 (3.7)	ns
Missed ps	10.8 (2.7)	11.7 (2.3)	8.6 (1.9)	ns
<i>At 0700 hours</i>				
Deleted letters p	55.5 (3.2)	57.4 (4.7)	59.4 (3.4)	ns
Missed ps	8.6 (2.0)	9.5 (1.5)	9.5 (1.5)	ns

ns, not significant.

Table 3. Mean difference (SEM) between Maddox Wing values at 0700 hours compared to control values at 2200 hours.

	Group A	Group B	Group C
Mean difference Maddox Wing values 2200–0700 hours	1.55 (0.3)*	1.85 (0.4)	3.45 (0.7)*

*p < 0.05.

The p-deletion test is a measure of concentration and manual dexterity. The ocular imbalance, an important variable for street fitness, is easy to perform, and measures the imbalance of the extra-ocular muscles. We found that some patients who were fully awake with practically normal p-deletion tests, were unaware of their existing strabismus.

It could be dangerous to drive a car if the ocular imbalance test is disturbed. It is almost impossible to compare hypnotic drugs on the basis of equivalent dose and to find any correlation between the patient's hypnotic or anxiolytic clinical response and plasma concentration.¹¹ All three hypnotics provided a normal sleep pattern, with significant differences in the subjective feelings of sleep sensation. The number of spontaneous awakenings was less with zopiclone, but no differences were found by any of the patients in the quality of sleep. Psychomotor performance as measured by Maddox Wing was more disturbed in group C (zopiclone). The p-deletion test showed no significant differences from control.

In a previous comparable study we compared oral flunitrazepam 2 mg and lormetazepam 2 mg and measured the Maddox Wing and p-deletion tests. In both tests there were changes from control values. The p-deletion test was not disturbed in this study, whereas the Maddox Wing test in the lormetazepam 1 mg and midazolam 15 mg group was less disturbed than the lormetazepam 2 mg group in the former study.

Different studies are considered, but the results of the lormetazepam groups for 1 and 2 mg may suggest a dose-dependent deterioration in psychomotor performance.

It was not possible to measure the time of total recovery (return to normal of the Maddox Wing values), because the patients subsequently received their premedication.

All three hypnotics, lormetazepam, midazolam and zopiclone provided satisfactory hypnotic effects. The results for midazolam, zopiclone and lormetazepam were comparable, except for significantly fewer spontaneous

awakenings in the zopiclone group. Psychomotor performance was less disturbed in the lormetazepam group.

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Purity of cylinder nitrous oxide

BOC specify a 97% minimum purity for nitrous oxide (N_2O), but it is expected that the normal concentration will be more than 99%. A decrease in nitrous oxide concentration of 4% was noted after a change of cylinders of N_2O used as a reference gas, and this prompted an analysis of the contents of a range of nitrous oxide cylinders.

Twenty-three cylinders of different ages, batches, and varying degrees of use were analysed by mass spectrometry. The concentration of nitrous oxide (AMU 30, 44) with respect to the purest cylinder available was recorded, together with the percentage of oxygen (AMU 32) and nitrogen (AMU 28) present. The purest cylinder was assumed to contain 100% N_2O , and the quantities of N_2 (9%) and O_2 (0.5%) in its analysis were assumed to result entirely from the breakdown of nitrous oxide in the mass spectrometer. The latter assumption, concerning O_2 as measured at AMU 32 is unlikely to be entirely true, since there should be little molecular oxygen from fragmentation of N_2O , but it should appear as atomic oxygen, AMU 16; this would indicate the presence of at least a small percentage of O_2 impurity even in our purest cylinder. The quantities of oxygen and nitrogen in the reference cylinder were subtracted from those in the remaining cylinders to determine the concentration of these gases initially present prior to the fragmentation of N_2O in the mass spectrometer.

The mean purity of the cylinders with respect to our 'reference' was 96.8% with a range of results of 6% (94–100%). The standard deviation of nitrous oxide concentration was 1.49%. Mean values of oxygen and nitrogen as impurities were 0.25% and 1.37% respectively.

There are several points that can be drawn from this analysis.

Not all nitrous oxide cylinders appeared to conform to the specified 97% minimum purity. Even assuming that our reference cylinder contained 100% N_2O , which is unlikely, one cylinder contained as little as 94% nitrous oxide, and the mean value for nitrous oxide concentration was 96.8%. Although we did not have a 100% pure N_2O cylinder to use as a Gold Standard reference, the fact that there was an unexpectedly large range of N_2O concentrations remains true and any impurities more likely to have been under rather than over-estimated.

The impurities present consisted mainly of nitrogen and oxygen, probably resulting from contamination with air. There remains a small difference, however, between the calculated total percent and 100%, suggesting the possibility of an additional, unidentified impurity.

The clinical significance of this is not immediately apparent, but problems may arise during the calibration of anaesthetic gas analysers if using a cylinder that is only 95% pure as a 100% reference for example. There may also

be some implications for low-flow, closed-system anaesthesia.

As a nitrous oxide cylinder empties, its N_2O concentration increases. Impurities such as nitrogen and oxygen are mainly present in the initial gaseous phase; the liquid phase contains purer N_2O . So for the purposes of calibration, a partly empty nitrous oxide cylinder is more likely to be accurate than a full one, the initial, impure gas having been cleared.

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S.L. SNOWDON
A.G. HEAD-RAPSON

A reply

This letter by Dr Snowden and Dr Head-Rapson highlights one of the phenomena of nitrous oxide with respect to the variation in purity of the gas and liquid phase. The nitrogen, which is produced in small amounts as an impurity in the manufacturing process, is controlled by venting gas from the production storage vessel so that the liquid purity is between 99.2% and 99.5%. The nitrogen, which is partially soluble in the liquid nitrous oxide, will always be in a higher proportion in the gas phase due to the relative volatility of the two gases.

Once the initial gas is drawn from the cylinder, further supplies are generated by boiling off liquid nitrous oxide. Nitrogen is more volatile than the nitrous oxide, so there is always a greater proportion of nitrogen in the gas phase. This has the effect of reducing the nitrogen in the liquid phase which, in turn, will improve the gas phase purity. When the cylinder is almost empty the purity of both phases approaches 99.9%.

The specification of the nitrous oxide is that the liquid phase purity shall be at least 99.0%. The purity in the gas phase will normally vary from 98.0% to 99.9% between a full and empty cylinder. We do not recommend the use of medical nitrous oxide for use as a calibration gas because of this variation. The most suitable certified cylinder is zero grade nitrous oxide, available from BOC Special Gases, which has a gas phase purity of at least 99.92%. If a certified cylinder is not available, we recommend using liquid phase sample from a medical cylinder since there is less fluctuation in the purity.

BOC Ltd,
The Priestley Centre,
Guildford,
Surrey GU2 5XY

P. HENRYS

Intrathecal diamorphine and multiple sclerosis

The case report by Leigh, Fearnely and Lupprian (*Anaesthesia* 1990; 45: 640–2) was interesting. Obviously the techniques described proved successful in the anaesthetic management of this case; however, the long-term effects of the chosen anaesthetic techniques on the relapse rate of the disease are the most important factors to be considered in patients with multiple sclerosis (MS) especially when the patient is so severely incapacitated.

It is always difficult to collect data on anaesthesia in comparatively rare diseases, and so it proves for anaes-

thesia and MS. Two studies implicate spinal anaesthesia in the exacerbation of MS.^{1,2} The retrospective study by Bamford, Sibley and Laguna suggested general anaesthesia had no effect on the relapse rate for MS (88 anaesthetics in 42 patients studied); however, a much smaller series of nine spinal anaesthetics in seven patients demonstrated one relapse in the month after the anaesthetic. The one relapse was after spinal anaesthesia for childbirth and the normal relapse rate for MS in the first 3 months postpartum has been shown to be three times as high as that in nonpreg-

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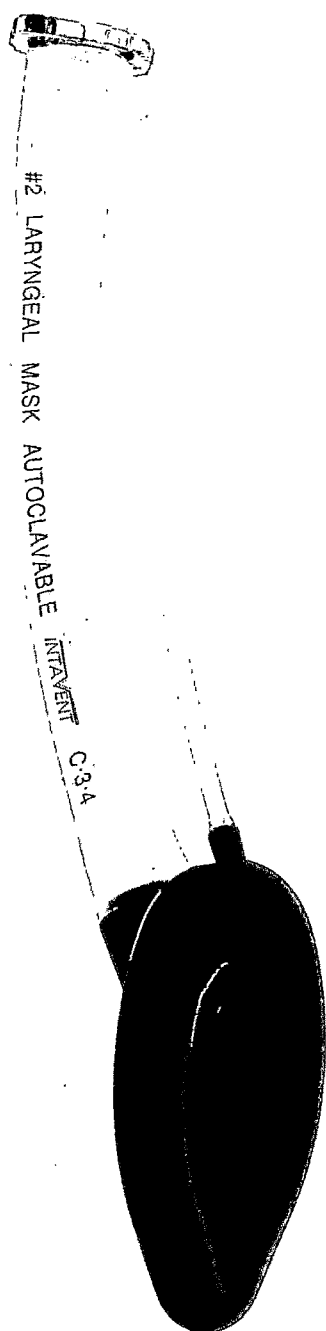
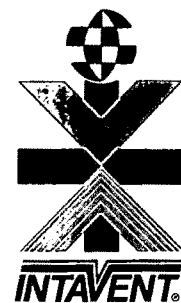
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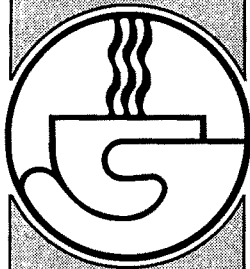
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nant patients³ so it is difficult to interpret this series. Bamford *et al.* also showed that local anaesthesia used for dental procedures did not appear to alter the normal relapse rate, which suggests that it is the site of placement that is important.

Stenuit and Marchand's² review of patients receiving spinal anaesthesia identified 19 patients with MS of whom two had definite aggravation of MS symptoms after spinal anaesthetics for general surgery.

More recently Bader *et al.*⁴ have reported their experiences with epidural local anaesthesia for childbirth in patients with multiple sclerosis, and found that epidural anaesthesia for vaginal delivery was not associated with a significantly higher incidence of relapses, when compared with those patients who received local infiltration of local anaesthetic for delivery. However, all of the women who experienced postpartum relapses had received concentrations of bupivacaine greater than 0.25%, suggesting to the authors that a higher concentration of drug over a longer period of time may adversely affect the relapse rate. They suggest that the relapses are due to the neurotoxic effects of the local anaesthetics which act on the demyelinated exposed area of the spinal cord.

Previous work^{5,6} has shown that the comparative concentrations of local anaesthetic in the white matter of the spinal cord during spinal and epidural anaesthesia is three to four times higher for spinal anaesthesia. Clearly further research is required on the usage of spinal anaesthesia in multiple sclerosis, but in the meantime it is perhaps unwise not to be cautious in the use of this technique for this condition, especially when general anaesthesia does not appear to be so controversial. The long-term possibility of relapses should, in my view, always take preference over short-term expediency, as the former is a further step downhill for the patient.

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A reply

My thanks to Dr Alderson for his interest in my article, and for the chance to reply to his letter. Dr Alderson concedes that spinal anaesthesia is not known to result in relapse of MS in the nonpregnant subject in the absence of postoperative pyrexia and agrees that general anaesthesia probably has no effect on the progress of the disease.

The decision that general anaesthesia was relatively contraindicated was a clinical one, reached after careful consideration of our patient as an individual. The work by Bader *et al.* although interesting, is not really germane to this discussion, since pregnant patients having epidural anaesthesia were studied.

Spinal anaesthesia does indeed merit re-evaluation in MS, and I hope that the technique (particularly if it includes intrathecal opioids) is used cautiously with consideration of risk and benefit in *all* patients.

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J. LEIGH

Ginger root, a new antiemetic

It was fascinating to read the article concerning the antiemetic effect of rhizome ginger (*Zingiber officinale*) for major gynaecological operations.¹ Even today rhizome ginger is still commonly used in Chinese herbal or folk medicine to reduce nausea and vomiting during pregnancy. The concoction is prepared by simmering plenty of peeled ginger with sweet malt vinegar for many hours. It is said that the younger the ginger the better the effect. Pregnant mothers drink it as often as they like to relieve the symptoms of nausea and vomiting. In fact, the use of rhizome ginger as a medicinal herb in China can be dated back many centuries. Traditionally it is used to reduce flatulence and other gastrointestinal symptoms, especially vomiting. In some compound prescriptions, it is also said to be effective in relieving muscular pain.² Rhizome ginger belongs to the *Zingiberaceae* family and contains 0.25-3% volatile oil, mainly zingiberol, zingiberene and camphorene.² The mechanism of action of ginger in reducing nausea and vomiting is unknown, but it is speculated that it probably works regionally on the gastrointestinal tract, rather than on the central nervous system.³

Dr Bone and her colleagues should be congratulated for their inspiration to conduct a clinical trial on this natural substance. When almost everything around us is turning green, perhaps it is time for anaesthetists to turn over a new leaf! After all, the most effective antiemetic agent may be hiding somewhere in our back garden.

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Exacerbation of a respiratory tract infection

Dr Campbell (*Anaesthesia* 1990; **45**: 561–2) describes a case in which a pre-existing mild respiratory tract infection became a life threatening illness during the course of an anaesthetic. The subsequent treatment of the patient seems commendable.

However, I disagree with the statement that 'it would be inappropriate to perform a chest X ray in all children with the symptoms and signs described in this case'. It is stated that the immediate chest X ray after operation changes were probably long standing and that a chest X ray beforehand might have provided useful information. This information would undoubtedly have altered the management. If surgery had still been deemed necessary, then being forewarned would certainly have led to different management throughout.

Clearly then, in a case where there are symptoms of illness, but no clinical signs to indicate its severity, a chest X ray is not in the least bit inappropriate but is in fact essential. Perhaps this case should serve to remind us that a respiratory tract infection is not always a benign illness and may have serious repercussions. Not every child with a runny nose needs an X ray, but a cough may be of more significance, and it is this group that probably ought to have a pre-operative chest X ray if surgery is required.

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A reply

Thank you for the opportunity to comment.

It is difficult to justify a chest X ray in all children with a runny nose and a dry cough. The patient we described appeared well, was afebrile, had neither tachypnoea, nor any clinical signs to suggest pulmonary disease. These negative findings would not indicate an X ray to be essential. Indeed, 10% of children with symptoms of an upper respiratory tract infection are exhibiting an allergic phenomenon. However, as mentioned in the case report, if cough is a prominent symptom, this investigation may be helpful.

It would have been useful to be forewarned of a chest infection. However, with the exception of antibiotic cover, this knowledge would not have altered peri-operative management significantly. Even in the light of the knowledge provided by a chest X ray beforehand, it is unlikely that the rapid deterioration in the patient's condition could have been predicted.

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Route for major vein cannulation

Powell and Beecher's case report (*Anaesthesia* 1990; **45**: 458–9) attests to the fact that insertion of central lines is not without hazard and has the potential for serious misadventure.^{1,2} The incidence of arterial puncture using the internal jugular route is around 0.5%³ and is 2–3% for subclavian approaches.^{3,4} We are aware of an unpublished death about 15 years ago when repeated attempts to cannulate the internal jugular vein damaged an atheromatous plaque on the carotid artery resulting in a fatal haemothorax.

Profound haemorrhage requiring repeated platelet transfusion and 2 hours of compression occurred in another case⁵ when arterial puncture was inadvertently performed in a patient with a platelet count of $20\,000 \times 10^9/\text{litre}$.

We have had a fatal case of laceration, at an identical site to the authors', after attempted subclavian vein cannulation. The patient later died, despite aggressive and successful attempts to control haemorrhage by our vascular and cardiothoracic surgeons from the effects of septicæmia, disseminated intravascular coagulopathy and haemorrhagic shock in the ITU.

All of these cases occurred with a catheter-over-needle system; it is time we abandoned this in favour of the safer Seldinger technique. The method⁶ is described for multiple entry into a single vein but has the potential for increasing the likelihood of complications, notably vein laceration, thrombosis and entanglement.⁷ Repeated punctures can only increase the chances of complications, however experienced the operator. Both methods are superseded by the use of multilumen catheters. These are of sufficient diameter to meet any clinical requirement. The additional cost has to be balanced by the greater safety of the catheter but would not account for more than 15% of the disposable bill for such a case.

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Drs Powell and Beechey recommend the use of the Seldinger technique to avoid this complication (*Anaesthesia* 1990; **45**: 458–9). However, just before their report was published I observed the same complication while using the Seldinger technique. A J-tipped metal guide wire was introduced without difficulty via the right internal jugular vein, and a 7-French gauge cannula for the insertion of a pulmonary artery catheter was advanced over this guide

wire. The introduction of this cannula is usually not without slight resistance because it is thick. This is possibly the reason why nothing abnormal was suspected. When after removal of the guide wire the aspiration of blood was impossible, the cannula was withdrawn until dark, non-pulsatile blood could be aspirated. The cannula was then reintroduced slowly, but aspiration of blood was again not possible.

There was a rapid decrease in blood pressure at this moment, which necessitated resuscitative measures, immediate sternotomy and the institution of cardiopulmonary bypass. The right hemithorax contained approximately 3 litres of blood. The bleeding was audible: pulsatile and hissing, and the right subclavian artery appeared to be damaged.

It is unlikely that the guide wire itself had perforated the vessel walls, but the use of a guide wire does not completely prevent the cannula from causing a perforation.

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C.D. PUNT

A reply

We endorse most of the comments made by Dr Ryan *et al.* about the use of multilumen catheters, but would like to add some points of our own.

We argue that the reduced risks of multilumen catheters arise firstly from the use of the Seldinger technique itself, and secondly from the avoidance of repeated vessel puncture.

The technique used for placement of multilumen catheters is really a refinement of the technique originally described by Seldinger.¹ Multilumen catheters are sited by first locating the vessel with a small needle, through which

a narrow gauge wire is threaded. A tunnel through the tissues of appropriate size is then created by use of a vessel dilator, and the cannula subsequently passed through the tunnel, over the guidewire. This is a fundamentally different approach from that employed in many 'Seldinger' single-lumen cannulation kits, where a large bore catheter is inserted over a large guidewire which has been placed through a large needle.

We do not believe that multilumen catheters are suitable for rapid, large volume infusion because of the length of the catheter and restricted diameter of even the largest lumen. Very large diameter cannulae can be inserted with relative ease using the modified Seldinger technique, and these probably represent the safest and most effective method of rapid resuscitation with massive transfusion. However, care is still needed; although we are not aware of any reports as yet, it can only be a matter of time before a pulmonary artery catheter introduction cannula is 'successfully' placed in the common carotid artery with dire consequences.

In Dr Punt's case it is difficult to envisage how the subclavian artery was damaged by the cannula if it had not previously been breached by the needle or guidewire. However, any technique for blind insertion of such large cannulae in this area cannot be totally safe. We do not propose that use of the Seldinger technique removes all risk, only that it is safer than a cannula-over-needle technique.

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A difficult cannulation of the right internal jugular vein

An 8-year-old male child presented for total correction of Tetralogy of Fallot.

Repeated attempts at cannulation of the right internal jugular vein (RIJV) after induction failed despite attempts through the high,¹ middle² and low³ approaches. Neither the vein nor the carotid artery was punctured during these attempts. Further attempts at RIJV cannulation were abandoned and the right subclavian vein was cannulated at the first attempt with a 16-G Secalon Seldy (Viggo, Swindon, UK) by the infraclavicular approach.

The surgeon noticed the absence of right superior vena cava (RSVC) and a persistent large left superior vena cava (LSVC) which drained into the large coronary sinus when the pericardium was opened. On opening the right atrium the surgeon had to cut off 5 cm of the central venous pressure cannula since it protruded into the surgical field.

The postoperative chest X ray demonstrated the cannula crossing the midline from the right side and going to LSVC.

Absence of RSVC with a persistent LSVC is a rare congenital anomaly⁴ with an incidence of 0.2%.⁵ This is the

first reported case to our knowledge of inability to cannulate RIJV due to absence of RSVC.

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Declaration of Helsinki

When a journal publishes articles or letters about a subject outside its own it is essential to ensure that the information published is factually correct; this is particularly the case when one enters the field of moral philosophy. A false belief is defined as a delusion.

Dr Grummitt (*Anaesthesia* 1990; 45: 496) is, in my opinion, incorrect in his interpretation of previous correspondence from Dr Drummond (*Anaesthesia* 1990; 45: 59). Dr Drummond does not comment in his letter on appropriate ethical standards because his letter does not describe the information published in the article to which he refers.¹ Patients who attended for pain relief therapy had procedures which resulted in pain relief, that is, the aim of therapy was accomplished. Drummond states that 'treatment was ineffective' but he should justify his assertion.

The correspondence has been published under the title 'Declaration of Helsinki' based upon Dr Drummond's assertion that there was some breach of this. If one reads this declaration there are contradictions between its subsections.² It is perhaps time that the whole declaration is properly reviewed and modified. If one believes so sincerely in a deontological basis to moral philosophy all anaesthetic research must be prohibited since our primary duty as anaesthetists is the safe care of our patients.

It is imperative, with the internal inconsistencies of the Declaration of Helsinki, that we review the entire concept of ethically acceptable standards in anaesthesia. The basic principles of moral philosophical debate are outlined elsewhere¹ and consist of issues of autonomy, beneficence, non-maleficence and justice. It is with the first of these that there is a significant dilemma in anaesthetic practice and research. How many patients actually understand what is the intended action of their anaesthetist in routine clinical practice? Their main concerns are, quite rightly, that they will become unconscious, that they will be safe and that they will recover. It is doubtful whether patients can be regarded as competent to consent to a routine anaesthetic, let alone to give consent to research during clinical anaesthesia. It is because of this that the nature and format of

ethical review must be stringent. The medical journals have a significant role to play in this.

The patient's autonomy is not threatened in some areas of clinical practice such as pain relief or local anaesthetic procedures. The patient is a free agent and can refuse the procedure and walk away. It is in such cases that the lesser principles of beneficence and non-maleficence and of justice become paramount. This is not to denigrate organised institutional ethical review which is of importance in these cases, in the case of research.

Our ethics have a strong deontological basis as much as in psychiatry. Our patients have rights and we our duties. Is there any place for utilitarianism?⁴ Is there any advantage for the specialty or the companies involved in the development of a new drug which outway the duty to our patients and allow the use of untried therapy? Can our specialty afford such paternalism? These are issues which need urgent and candid debate. If as a specialty we do not sort out these problems, and soon, then they will ultimately be dictated to us from our patients and by our employers. The recent legislation embodied in the Human Fertilisation and Embryology Act (HMSO 1990) now enshrines in law compulsory ethical review of clinical practice, in addition to clinical research. It is not unreasonable to assume that such compulsory ethical review of clinical anaesthetic practice may follow at some stage.

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Atropine during halothane anaesthesia for children

Atropine is routinely administered to prevent or to antagonise the halothane-induced bradycardia in children. Relatively large intravenous doses of atropine (0.02 mg/kg to 0.03 mg/kg) are recommended.^{1,2}

Do we need these higher doses in routine mask cases in children? Thirty-two ASA 1-2 children (3-10 years) for minor surgery were the subjects of the study. Institutional approval and parental consent were obtained. The children received no premedication, and anaesthesia was induced by

inhalation of halothane in nitrous oxide and oxygen (2:1). When the end-expiratory halothane concentration had reached to 1.5%, intravenous atropine 0.01 mg/kg (Group A, $n = 17$), or 0.02 mg/kg (Group B, $n = 15$) was administered. Halothane concentration was kept constant for 10 minutes after administration of atropine. Pulse rates were measured by Dinamap 1846 monitor (Critikon) before the induction, before atropine, and every minute for 10 minutes after atropine administration. Student's t -test was

Table 1. Mean (SD) pulse rate changes after atropine administration.

	Awake	Halothane 1.5%	Time after atropine (minutes)									
			1	2	3	4	5	6	7	8	9	10
Group A	106 (18)	84 (15)	126 (14)	136 (9)	138 (10)	140 (10)	141 (10)	142 (10)	142 (9)	142 (9)	142 (10)	142 (10)
Group B	98 (12)	81 (11)	121 (14)	137 (9)	139 (8)	141 (6)	142 (9)	143 (8)	144 (8)	144 (9)	144 (8)	144 (8)

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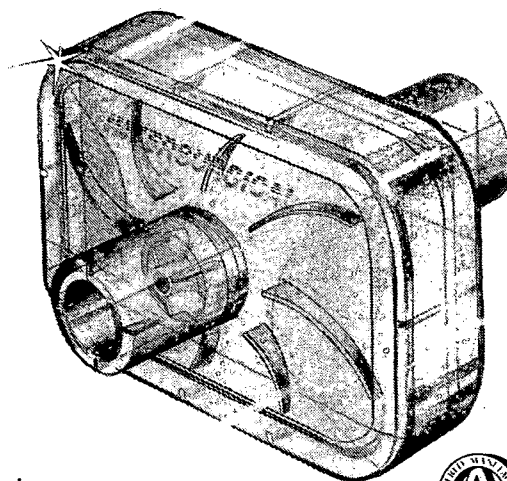
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Edited by

Felicity Reynolds, *Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.*

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utilised for statistical analysis and a *p* value less than 0.05 was considered significant. Age and weight were comparable in two groups.

The pulse rate decreased to about 80% of pre-induction pulse rate after halothane induction, but increased to 140% of pre-induction rate after atropine administration in both groups, and there were no differences between the groups (Table 1).

Intravenous atropine 0.01 mg/kg increased the halothane-induced slowed pulse rate more than the rate in awake state. Do we need to increase the pulse rate during halothane anaesthesia more than the rate in the awake state? Since halothane-induced myocardial depression can only be at most partially improved by vagolysis,³ the higher dose of atropine seemed unnecessary, at least when the airway is maintained by mask.

Hyoscine derivatives in children

A 14-month-old boy who weighed 8.4 kg presented for endoscopic retrograde cholangiopancreatography to be carried out in the X ray department under general anaesthesia. He was alert and fit apart from unexplained jaundice of hepatobiliary origin. Investigations were all within normal limits apart from these liver function tests: bilirubin 100 µmol/litre, alkaline phosphatase 1732 units/litre, aspartate transaminase 1496 units/litre, alanine transaminase 1210 units/litre and albumin 33 g/litre.

Anaesthesia was induced with Entonox and isoflurane. Atracurium 0.6 mg/kg was injected through an indwelling 22-gauge cannula and a plain 4.0-mm tracheal tube inserted. Maintenance of anaesthesia was with Entonox, isoflurane 0.5–1.0%; atracurium 2 mg was given as required. Ventilation of the lungs was by hand through a Jackson-Rees T-piece. Difficulty with cannulation of the common bile duct was experienced during the operation and, without knowledge of the writer 10 mg of hyoscine n butyl bromide (Buscopan) was administered intravenously by the endoscopist to relax the sphincter of Oddi. The procedure was abandoned after approximately one hour and percutaneous transhepatic cholangiography was performed without difficulty. Subsequently the patient's lungs were ventilated with oxygen and residual neuromuscular paralysis was reversed with neostigmine 80 µg/kg and atropine 20 µg/kg. Spontaneous ventilation was promptly restored. The child remained extremely sleepy, disorientated, had a flushed appearance, dilated pupils, dry mouth and Cheyne Stokes type breathing. Seventy minutes

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elapsed before he was awake and able to make active efforts to remove the orotracheal tube. The delayed recovery was thought to be due to hyoscine for two reasons. The clinical signs were compatible with hyoscine overdosage and the anaesthetic agents employed are short acting. The endoscopist confirmed that Buscopan was given only if cannulation of the common bile duct proved difficult; the dosage was approximately 1 mg/kg.

The case has several important implications.

Ten milligrams (approximately 1.25 mg/kg) of hyoscine hydrobromide is an excessive dose compared with the (adult) recommended dosage of 10 mg initially, repeated in 30 minutes time (if necessary) to a maximum of 20 mg (up to 0.25 mg/kg) as indicated by the manufacturers, Boehringer Ingelheim Ltd.

Anaesthetists must have knowledge of the nature and dosage of drugs which are given by other doctors during operative procedures and should be informed personally before their administration since such drugs may have a profound effect upon the anaesthetic.

Hyoscine is best avoided in persons under one year or over 65 years of age and alternative methods of relaxation of the sphincter of Oddi should be considered in these age groups.

Anaesthetists need to be vigilant at all times and particularly when other drugs are administered.

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Transient blindness

We read with interest the recent case report about loss of vision after transurethral resection of the prostate (TURP).¹ A similar case of glycine absorption has recently occurred in our hospital.

Our case had some features that do not appear to have received much attention.

A previously healthy 72-year-old man was undergoing transurethral resection of a large prostate under spinal anaesthesia. Towards the end of the operation he became restless with a bradycardia and hypotension. He responded somewhat to blood transfusion and ephedrine; however, after an hour in the recovery room, he became progressively more drowsy. He was admitted to the ICU where his cardiovascular system was stable but started spontaneous twitching movements, and showed a positive Chvostek's sign. Hypertonic saline was started but he had a generalised convulsion and his trachea was intubated and his lungs ventilated. His fluid management and biochemical results are shown in Table 1. His pupils were fixed and dilated for

12 hours, but he had a gag reflex and no other signs of brainstem dysfunction. He started to wake up and his trachea was extubated 40 hours after operation and he made an uneventful recovery.

Firstly, the biochemical results suggested the presence of 10–20 mmol/litre of an anion that caused a metabolic acidosis and severe disproportionate hypocalcaemia. The major metabolites of glycine are serine and glyoxylic acid, which are in turn metabolised to pyruvate and oxalic or formic acid. Hyperoxaluria is reported to occur after TURP and it seems likely that this patient suffered some degree of hyperoxalaemia. We suggest that in the treatment of a cardiovascular emergency in a TURP, the possibility of hypocalcaemia be remembered.

The presence of unreactive pupils supports the contention that high levels of glycine have direct inhibitory effect on the retina as occurred in the case reported by Russell.

It is interesting to speculate on the reason for the 'lucid interval' between the absorption of the glycine and the

Table 1. Biochemistry.

	Before operation	Hours after operation						
		3	6	9	12	18	24	48
Na (mmol/litre)	142	115	120	124	124	128	136	136
K (mmol/litre)	5.2	5.6	4.8	4.0	3.6	4.0	3.7	3.2
Urea (mmol/litre)	7.4	10.6	12.0	12.9	15.4	18.5	12.1	7.3
Ca (mmol/litre)	—	1.47	—	1.78	1.92	1.83	2.12	2.23
Albumin	—	29	—	30	30	29	33	38
Mg (mmol/litre)	—	—	—	—	0.60	—	—	0.83
PO ₄ (mmol/litre)	—	1.05	—	—	0.98	—	—	0.80
Hb g/litre	119	110	101	98	—	83	—	108
Osmolality	—	284 mosm/kg						
NH ₃	—	—	44 µmol/litre					
Bicarbonate	—	11	12	12	14	18	17	19
Osmolar gap = 22 mosm/kg								
Treatment								
Blood	4 U							2 U
3% saline				300 cc	—	500 cc		
Mannitol 15%			200 cc					
Ca gluconate			1 g		1 g	1 g		

occurrence of the cerebral disturbance. The build up of a toxic metabolite seems to be the most plausible explanation. Others have measured high concentrations of ammonia,² but this did not occur in our case, which would suggest that one of the other metabolites may have been involved. Perhaps this compound may be responsible also for the postoperative confusion that is not uncommon in these patients.

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A reply

Thank you for the opportunity to comment.

Desmond¹ states that there are probably very few operations in which a patient can be subjected simultaneously to such a large number of unphysiological insults as in a transurethral prostatectomy (TURP). Drs Sleigh and Miller witnessed a severe case of the TURP syndrome.² So many biochemical and pathophysiological abnormalities are present during a full blown TURP reaction that the precise aetiology of all the signs and symptoms remains unclear.

Hahn³ discusses the metabolic effects of glycine, and describes a substantial increase in the aggregate concentra-

tions of nonessential amino acids other than glycine and serine, probably as a result of glycine metabolism.

Charlton,⁴ in his case of cardiovascular collapse associated with transurethral prostatectomy, observed a dramatic response to intravenous calcium, and suggested that an acute reduction in plasma calcium concentration may have been present, an assumption now supported by the preceding report.

My patient's pupillary responses were unfortunately unreliable as a result of the effects of recent surgery and topical drugs; however, it is interesting that Drs Sleigh and Miller noted fixed and dilated pupils without other signs of brainstem dysfunction.

Finally, in my patient the confusion, agitation and amnesia began halfway through the resection, and, if anything, began to improve about an hour later.

One wonders if perhaps the absorbed glycine is handled differently by different patients, depending as well on the amount absorbed.

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Cold statistics

McCrirrick and Hunter (*Anaesthesia* 1990; **45**: 443–4) recently studied the effect of propofol temperature on the pain experienced by patients.

In the study 71 patients were divided into two groups; 34 patients in group 1 received propofol at 4–5°C, and 37

patients in group 2 received propofol at room temperature. The pain experienced by each patient was assessed on a 0 to 3 scale ranging from no pain (0) to severe pain (3). The results are summarised in their Table 2.

The overall incidence of pain in group 2 was 46% and in

group 1 23%. McCrirrick and Hunter claim that this is significantly different with $p < 0.05$. A Chi-squared test of homogeneity with Yates' continuity correction² gives a nonsignificant result ($p = 0.08$). Excluding the continuity correction the significance level becomes $p = 0.048$ (i.e. $p < 0.05$). It is well-known that inclusion of the continuity correction greatly improves the validity of the Chi-squared test in this 2×2 situation, here comparing the proportions of cases with pain levels 1+2+3 and level 0 in the two groups.

Rather than combine pain levels 1, 2 and 3 together a Chi-squared test of homogeneity can be performed on the 2×4 data set of their Table 2. This gives $p = 0.095$. To overcome fears that the Chi-squared test may not be appropriate in this small sample case a randomisation test^{1,2} was carried out. Ten thousand simulations gave $p = 0.090$ (SD 0.003). No significant difference is observed with these data.

A similar randomisation test for the 2×2 data case described above yielded $p = 0.051$ (SD 0.002) which confirmed the nonsignificance of the Chi-square test result.

The only significant result is that comparing the proportions of severe pain, level 3, and nonsevere pain, levels 0+1+2, in the two groups. A Chi-squared test with continuity correction gave $p = 0.045$. Fisher's exact 2×2 test³ gave $p = 0.019$, and a randomisation test with 10 000 simulations gave $p = 0.020$ (SD 0.001). The result quoted in the paper was $p < 0.025$.

Injection of propofol at 4–5°C significantly reduced the incidence of severe pain experienced by patients, but more data would be needed to assess the effect of low temperature on the incidence of mild or moderate pain.

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A reply

We are grateful for Dr Baczkowski's comments on our study report. They reflect an academic controversy that bedevils the subject of analysing 2×2 contingency tables. Bland noted this long-standing controversy continues to generate as much heat as light.¹ We are ill-equipped to enter into the affray; we adhered to common practice in our choice of test strategy. We used the uncorrected Chi-squared test statistic when all of the cell expectations in the 2×2 table exceeded 5, and the Yates' corrected

Chi-squared test statistic, supported by Fisher's Exact test, when this condition did not obtain.

Following that strategy to the letter our study conclusions stand: that the use of cold propofol significantly reduced pain on injection ($p < 0.05$). The use of the corrected Chi-squared statistic by Dr Baczkowski to assess the difference in the overall incidence of pain between the two study groups (levels 1+2+3 compared with level 0) is not indicated under our test strategy and the fact that this delivers a nonsignificant test outcome ($p = 0.08$) appears to us to be rather more a problem for the statistical community than to the present authors. Dr Baczkowski will be aware, although he does not tell us as much, that statisticians do not speak with one voice in respect of the virtues of the corrected Chi-squared statistic; some authorities hold the view that its use is inadmissible under any circumstances whatsoever, which is a view reflected in the texts of Pocock² and Fienberg.³

We are in no position to argue the technical merits, or lack of them, in our chosen test strategy. We have been guided by the authority of Bland (*op. cit.*, p. 245) in respect of the conditions required for valid application of the uncorrected Chi-squared test. If Dr Baczkowski takes issue with that choice, he should perhaps address himself to the authors of policy, not its servants.

The Fisher's exact test probability ($p = 0.019$) obtained by Dr Baczkowski in respect of the severe pain versus nonsevere pain comparison is incorrect. The two-sided test probability relevant to the present discussion is $p = 0.039$ (following Yates⁴) as Dr Baczkowski will, on reflection, no doubt confirm. The probability $p < 0.025$ cited in our paper should have been $p < 0.05$.

Our audience would have been better served, with the wisdom of hindsight, had we ensured that an account of the precise details of our test strategy had survived the editorial process. Better still, had we elected to use confidence intervals on the population differences in the pain response proportions, these would not have altered our study conclusions but they would have clearly represented the uncertainties implicit in our (or any other) study data.

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Bimanual cricoid pressure

The illustration in the letter by Drs Crowley and Giesecke (*Anaesthesia* 1990; 45: 588–9) does indeed show the technique for application of bimanual cricoid pressure. However, it also clearly shows the occiput resting at the same level as the shoulders, which I would maintain is certainly not a good 'sniffing' position.

Cricoid pressure is used mainly during emergency rapid sequence induction, so optimal positioning for intubation is

essential, before induction of anaesthesia is commenced. Anterior flexion of the lower cervical spine, by raising the head on a firm pillow, still allows extension of the remaining cervical vertebrae and with it effective cricoid pressure. More importantly, it improves the alignment of the pharyngeal and laryngeal axes, thus allowing easier visualisation of the glottis and with it tracheal intubation.^{1,2}

Cricoid pressure is, in my experience, infrequently performed bimanually. This may be because it is not taught in this way or more likely that the assistant's other hand is usually required during the process of cuff inflation. Failure of cricoid pressure, however, seems more likely to result from too little force applied or inappropriate release, rather than the absolute technique of application.^{3,4}

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Correct placement of tracheal tubes

The need to confirm correct placement of tracheal tubes is mandatory; Dr Higgin's suggestion (*Anaesthesia* 1990; 45: 591) that the Fenem CO₂ detector be used for rapid confirmation (in children) surely ignores the undisputed evidence of Andersen and Hald.¹ Auscultation of not only both axillae but also the epigastrium gives confirmation of correct placement in 100% of cases, and at no added cost. Auscultation of the lungs alone can result in a wrong conclusion in 15% of cases when the oesophagus is ventilated.

The Fenem CO₂ detector (at £13.60 per unit) has a limited life-span, and used as suggested, would need to be replaced daily in any one theatre. A significantly cheaper and reusable oesophageal detector is already available.²

The Fenem CO₂ detector device

Dr Denman *et al.* (*Anaesthesia* 1990; 45: 465-7) state that we reported a failure of the oesophageal detector device (ODD).¹ We did not. We reported a false-positive: a bulb that did not refill when the tube was in the trachea. A failure to detect the oesophagus, an instant bulb refill with the tube in the oesophagus, has not been reported. False-positives are not dangerous, since they merely provoke extra interest in the position of the tube.

Dr Lunn has pointed out that a substantial reduction in the mortality due to oesophageal intubation may only be achieved by testing every patient.² We believe that every patient could be tested with the ODD as described by Williams and Nunn.³ The device is easy to use, lasts more-or-less forever and costs £3.08 (one Ellik bulb, GU Ltd). Failure is not reported. However, such a policy would be expensive if the Fenem detector were used: it is marketed as a single-use device at £13.75.

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A reply

Thank you for the opportunity to reply. We are grateful for the clarification that it provides in explaining that they obtained a false-positive as opposed to a false-negative result with the detector.

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However, the stethoscope has existed for many centuries and with correct usage should last a lifetime.¹

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We do not agree that false-positives are not dangerous. If the bulb of the ODD does not refill when the tube is correctly positioned, then sufficient doubt may be engendered in the mind of the stressed anaesthetist over the position of the tube that they may remove it. 'When in doubt take it out' remains an excellent axiom.

We still believe this represents a failure of the ODD but this does not detract from its usefulness at times. No single device is the panacea for all situations and anaesthetists must keep an open mind and understand the advantages and disadvantages of the equipment they use.

The FEF detector is expensive but it remains a singularly useful piece of apparatus during difficult intubations. The rapid breath-by-breath colour changes which illustrate the presence or absence of carbon dioxide in the respiratory gases are most reassuring and we believe are unique.

It must be stressed that we do not advocate that any anaesthetist should rely totally on one piece of apparatus for all situations, but we do suggest that the Fenem CO₂ detector is a useful addition to our monitoring armamentarium.

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D.J. WILKINSON
M. HAYES
D. HIGGINS

This is a response to Drs Wilkinson, Denman and Higgins' letter on the Fenem FEF CO₂ detector (*Anaesthesia* 1990; 45: 694). Drs Wilkinson and his colleagues seem to have responded to my comments on the FEF CO₂ detector (*Anaesthesia* 1990; 45: 251) at a personal level and this is disappointing. However, I would like to assure them that some of my comments were based on doubts expressed by other anaesthetists about the potential fallibility of end-tidal CO₂ measurements for the detection of oesophageal

intubation.^{1,2} Dr Wilkinson and his colleagues' comment that it is vital in anaesthetic practice to keep an open mind is absolutely correct and it is why we all welcome constructive comments on any new device or practices introduced into anaesthetic practice, particularly if the topic is on safety.

Is it safe to ventilate a patient's lungs for six breaths through the FEF CO₂ detector before conclusions are drawn about the exact location of the tube? If the tube was located in the oesophagus, particularly in a patient with a high risk of aspiration, subsequent dilatation of the stomach may increase the risk. The oesophageal detector device (ODD) is best used before ventilation after intubation to check tube position.

There is an inaccuracy cited by Dr Wilkinson and his colleagues about the reported failure of the ODD. They did not note that the failure reported by Dr Calder *et al.* (*Anaesthesia* 1989; 44: 705) was with Dr Nunn's modification of the ODD (ODD- Mark II), using Ellick's bulb as a means of suction and *not* the original ODD with which it is possible to reduce the strong suction effect by a slow constant aspiration of the plunger. The strong suction effect may cause a false-negative result (negative pressure with the tube placed in the trachea).

The difference between the ODD Mark I and II were discussed before in relation to avoidance of a false-negative result (*Anaesthesia* 1989; 44: 930-1). Furthermore, a low

false-negative rate is deemed not clinically significant according to the criteria for an ideal test for detecting oesophageal intubation³ and therefore should not necessarily be reported as a failure of the test. A recent prospective comparative trial⁴ of five simple and commonly used methods have confirmed the reliability of the ODD Mark I.

It seems prudent, in the absence of direct visualisation of the passage of the tube through the trachea, to use a number of tests in combination with a high index of suspicion to avoid the preventable error of oesophageal intubation.

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Ohmeda Oxicap 4700

This is a report of a potentially serious hazard.

A pulse oximeter functioned incorrectly during an operation. The procedure when the oximeter was set up is unclear since two anaesthetists and an ODA were involved. The pulse oximeter showed a pulse rate of 100/minute, a saturation of 98%, and the plethysmographic trace indicated a strong signal whether or not it was attached to the patient. The capnometer functioned normally.

The implications of this malfunction are obvious.

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F.B. DUNCAN

A reply

Thank you for the opportunity to reply and comment.

The condition that Dr Duncan describes on the Ohmeda 4700 Oxicap was identified and traced to the software.

On start-up there is a possibility that the calibration signal of SpO₂ 98% and pulse rate 100 beats/minute can become 'locked' onto the monitor display screen. This 'lock-up' condition can only occur during the 'power-up' sequence when the device is first turned on and remains until the unit is turned off.

A letter was circulated to all customers which clearly explains the nature of this condition and a 'software upgrade' has been developed. We are contacting customers to arrange fitting this modification as soon as possible.

Users should in the interim verify correct operation after the start-up procedure by ensuring that 'Probe off patient' is displayed on the monitor when it is not connected to a patient, and that when in use the waveform and signal strength indicator show a physiological response.

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R. THUILLIER

A faulty monitor

A potentially lethal fault has occurred in a Datascope 2200I monitor.

A fit, 34-year-old woman presented for local resection of a choroidal melanoma under profound hypotensive anaesthesia. She received our standard anaesthetic of thiopentone, curare, nitrous oxide, oxygen and enflurane. Monitoring included an ECG and invasive arterial pressure displayed on a Datascope 2200I. Twenty minutes after induction her arterial pressure was 98/55 mmHg and we began an infusion of trimetaphan and sodium nitroprusside to decrease her mean arterial pressure to our target of 30 mmHg. Five minutes later, despite a high infusion rate, the arterial pressure remained at 98/55 mmHg with an unchanged waveform. We wondered if the zero had drifted and opened the transducer to air. The arterial trace showed a flat line at zero but the monitor continued its digital

display of 98/55 mmHg. We re-zeroed the transducer to see what would happen and the digital display returned to zero. The transducer was then switched back to the patient and the monitor showed a damped arterial trace consistent with the displayed pressure of 43/22 mmHg. The monitor showed appropriate waveforms and pressures during the rest of the anaesthetic and the patient made an uneventful recovery.

The implications are serious. Many modern monitors display an analogue signal from which the digital readings are derived. An anaesthetist can thus assess the quality of the signal and make appropriate decisions about the credibility of the digital display. When a monitor displays incorrect analogue and digital data that are entirely consistent with each other an anaesthetist will not readily disbelieve them. It seems that our Datascope 2200I con-

tinuously replayed the same screen of information and did not display the patient's actual arterial pressure. There was no discontinuity in the arterial waveform to suggest that this was happening and the ECG continued normal throughout.

We were lucky that we were deliberately manipulating this patient's blood pressure and realised fairly quickly that something was amiss. In other circumstances we might well have congratulated ourselves on our stable anaesthetic and been unaware of life-threatening changes in our fully monitored patient.

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D.L. PAUL
J.G. TODD

A reply

We believe that, as far as Datascope can ascertain from all information received to date, the problems experienced with the 2200I Invasive Pressure Trace are batch-related. We have had no similar reports with any other 2200Is worldwide. All 2200Is from the batch in question have been removed from customer sites and returned to our corporate facility in Paramus, New Jersey.

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C. ELDRED

A Brain laryngeal mask airway connector

The Brain laryngeal mask airway is a versatile invention that provides an intermediate between mask anaesthesia and tracheal intubation. Its use is described in an increasing number of clinical settings; however, owing to the length and natural curve of the tube portion of the airway, problems with connexion to the breathing system away from the surgical field may arise. We have also found that if the breathing system is connected to the airway over the forehead, it may become displaced. By joining two standard Portex swivel connectors, as shown in the Figure, the problems described can be overcome. They have the advantage of being readily available, designed for the task, have a small deadspace (13 ml), and a universal swivel; by glueing them together, the risk of disconnection is reduced.

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M.M. CROSSE
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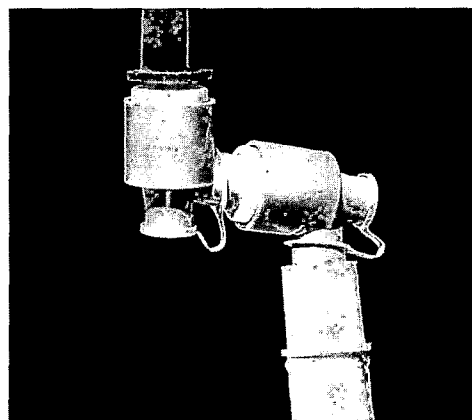


Fig. 1.

Laryngeal mask airway for Caesarean section

We have been following the papers, and correspondence, about the use of Brain's laryngeal airway with interest. We are, however, alarmed at reports of the use of this device for obstetric anaesthesia.

Imagine that you are about to undergo Caesarean section under general anaesthesia and that you will be a failed intubation. The latest triennial report on maternal mortality¹ gives a fairly clear indication of your likely circumstances. You will probably be obese, and undergoing an emergency section. Your anaesthetist will be a trainee who is inexperienced and unsupervised.

The report is not clear as to whether your anaesthetist reads the journals.²⁻⁴ If he (she) does, he (she) is likely to abandon any failed intubation drill and reach for a Brain's laryngeal airway.⁵ We suggest that you now face one of three possible outcomes.

Firstly, if you are inadequately anaesthetised you will cough, gag, breath-hold, and probably develop laryngospasm.⁶⁻⁸ You will then almost certainly die.¹

Secondly, placement may not be possible, in which case you are worse off because you have been exposed to the risks of the first option, and have had time wasted. You will probably die.

Thirdly, if you are deep, hypoxic, or already dead, laryngeal airway placement may be possible. To keep it in place however, anaesthesia will have to be sufficient to obtund laryngeal reflexes. You will therefore be unable to protect yourself from aspiration. The laryngeal airway also does not afford you any protection,^{5,9,10} your tracheal toilet

therefore being reliant upon cricoid pressure, if still applied. Your outcome in these circumstances therefore depends upon whether, as they say in the cinema, you feel lucky.

Eight maternal deaths in the last triennial report were attributed directly to difficult tracheal intubation. There is little doubt that the laryngeal airway is an excellent difficult intubation tool.^{11,12} Had it been available, and had it been used as a last resort in the hypoxic, it might have saved some of these lives. Used inappropriately however, it has the potential to turn eight difficult intubation deaths into eight deaths from aspiration. We suggest that the profession thinks carefully about the use of the laryngeal airway for difficult emergency obstetric intubation by junior anaesthetists.

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A reply

Our publication about the use of the laryngeal mask for the impossible tracheal intubation during obstetric anaesthesia has provoked considerable debate. The disadvantages and dangers outlined in this letter are valid, but in our opinion the laryngeal mask can still be a useful item on the 'difficult intubation' trolley.

Our main considerations in the management of an impossible intubation are unchanged and are to accept that intubation is impossible after no more than two or three attempts, to maintain oxygenation and cricoid pressure; and to allow the patient to wake up, if appropriate. These points are stressed in the teaching of junior anaesthetists.

Adequate oxygenation of the unconscious patient with a conventional facemask may not be possible, particularly in an obese obstetric patient. The laryngeal mask may then be a very useful alternative; cricoid pressure and the left lateral position are maintained until laryngeal reflexes return. It may, however, be justifiable to continue general anaesthesia via the laryngeal mask, by manual and then spontaneous ventilation, provided airway patency is satisfactory, and cricoid pressure is maintained. In the case we described, persistent fetal bradycardia was noted before induction of anaesthesia, and the resumption of spontaneous ventilation was delayed.

We heartily recommend that anaesthetists, both junior and senior, gain experience in the use of the laryngeal mask in elective nonobstetric anaesthesia, before attempting to use it in an emergency. This applies to most forms of equipment for 'difficult intubations'.

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Standard colours for cannulae

I applaud Tordoff and Sweeney's report which draws attention to the lack of any standard colour coding system for intravenous cannulae and the confusion this produces (*Anaesthesia* 1990; **45**: 399–400).

Contrary to their assertion, the ISO Standard 6009 on colours for identification of hypodermic needles covers all sizes from 12 gauge to 26 gauge. This may not be clear on initial examination of the standard because the sizes are given in the outside diameter in millimetres. It would thus be possible to use the colours in this existing ISO standard for intravenous cannulae also. It is important to achieve agreement before 1992, otherwise the CEN European Standards Secretariat may be instructed to produce a standard for Pan-European application which may not be to everyone's liking. Many manufacturers already comply with the French standard, so these colours have been proposed by ISO Committee TC84/SCI/WGI (see Table 1) for adoption in their ISO draft standard Document N14.

This initial Document N14 requires the following to be

marked on the unit package: outside diameter in mm, gauge size optional; the length of the cannula; the flow rate in mm/minute using British Standard 48 43:1987, appendix B test; the minimum inside diameter; and the colour of the hub.

It is important that the colour patch and size number on the package be large so that in an emergency the correct size of cannulae can be identified promptly, say at 1 metre in 215 lux lighting (minimal hospital corridor lighting). (See Wallace Y-Can 23 g and Deseret Novalon 20 g for excellent examples).

Pale pastel shades of beige, grey, green and pink should be avoided since 8% males find it difficult to distinguish between pastel colours due to deficient colour vision.

This ISO standard is currently under development so UK users who wish to provide clinical input should contact Mr David Upstone, BSI, 2 Park St, London W1A 2BS, tel: 071-629-9000, the Secretary TC 84/SCI; or Mr C.G. Grenshaw, Smith Industries Medical Systems, Portex

Table 1. ISO TC84 colours, ISO 6009 colours and present manufacturers' colours (USA).

Sizes	Sizes (gauge)				
	14	16	18	20	22
TC84 colour	Orange	Grey	Green	Pink	Blue
US makers	Medicut	Abbott	Abbott	Abbott	Abbott
using these	Cathlon	Medicut	Medicut	Medicut	Medicut
colours	Vicra	Cathlon	Cathlon	Cathlon	Angiocath
		Vicra	Vicra	Vicra	Cathlon
					Vicra
Use of other colours					
Abbott	Gold				
IV Cath (BD)	Light grey	IV Cath lavender	IV Cath pink	IV Cath yellow	IV Cath grey
Longwell (BD)	Olive	Longwell purple	Longwell pink	Longwell yellow	Longwell black
Angiocath (Deseret)	Pink	Angiocath yellow	Angiocath tan	Angiocath green	
ISO 6009 colours	Pale green	White	Pink	Yellow	Black

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BS4843 does not specify colour coding because of its complexity. Historically, colour coding of intravenous cannulae has followed the brand leader at a particular time in each country; a further confusion is the United States' misnomer: catheter. The confusion in colour coding is worldwide.

The solution would be for European and US Health Authorities to direct, in unison, that for 2 years intravenous cannulae hubs shall be uncoloured (natural) and an agreed international colour coding should appear only on the package. During these 2 years both manufacturers and hospitals can clear-through their inventories ready for the international coding to be applied after the 2-year period. If colour coding is introduced without such a process there will be even more confusion.

Our industry agrees that the present situation is unsatisfactory and would welcome a solution.

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Unusual neurological sequelae

The article by Drs Saunders and Harris (*Anaesthesia* 1990; 45: 552-7) was interesting since I have recently had a similar experience.

A fit, 23-year-old, rather anxious man, presented for excision of four wisdom teeth under general anaesthesia as a day case. He was unpremedicated. Anaesthesia was induced with propofol 160 mg (2.5 mg/kg) after alfentanil 1 mg and lignocaine 20 mg. Tracheal intubation was facilitated with vecuronium 3 mg and ventilation of the lungs was with 5% enflurane for 30 seconds. Anaesthesia was maintained with nitrous oxide, oxygen (30%) and enflurane (2%); ventilation was assisted by hand until adequate spontaneous ventilation returned. The operation lasted 25 minutes. Analgesia afterwards was provided by subperiosteal injections of bupivacaine 0.75% (6 ml).

Recovery was slow; the patient remained asleep or drowsy for over 2 hours. He complained of a 'burning sensation' inside himself but the axillary temperature remained at 36.5°C. His conscious level alternated between somnolence, from which he was easily rousable, by squeezing his hand or by oral suction, and light sleep. He suffered several apnoeic episodes greater than 30 seconds duration during the periods of sleep; these were unresponsive to naloxone but were abolished by 40 mg doxapram intravenously. Two doses were given at half-hour intervals. The oxygen saturation was above 90% and he breathed oxygen 4 litres/minute from a Hudson mask. Return to full consciousness took about 3 hours.

Initial neurological examination revealed slight weakness of the left arm, particularly reduced grip strength, although tone and tendon reflexes were considered to be normal. There were no other focal signs.

The weakness resolved as a normal level of consciousness returned and on neurological examination by a physician 5 hours after the anaesthetic, no abnormality was detected.

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S.J. MATHER

We read with interest the excellent article by Saunders and Harris (*Anaesthesia* 1990; 45: 552-7) describing unusual neurological sequelae after propofol anaesthesia, and

would like to report a similar case. A previously fit, 21-year-old man presented as an emergency for exploration of an injured wrist. He was unpremedicated and at induction was noted to be very anxious. Anaesthesia was induced with propofol and vecuronium, and his trachea was intubated and his lungs ventilated with oxygen, nitrous oxide and isoflurane. Ten milligrams of papaveretum were given intramuscularly for analgesia. The operation lasted 30 minutes. Muscle relaxation was reversed with neostigmine and glycopyrronium, his trachea was extubated and he was admitted to recovery where he spoke to the anaesthetic nurse. However, suddenly, his limbs became rigid and he lapsed into unconsciousness. He became totally unresponsive to deep pain, developed a curious, regular hiccough, like a diaphragmatic spasm, associated with a backward jerk of the head, which occurred about six times a minute.

His breathing was normal between these episodes and he maintained an oxygen saturation of 99%.

His pupils appeared normal and there were no localising signs. He had increased muscle tone in all four limbs and was hyper-reflexic. Plantar responses were normal and there was no ankle clonus. Blood glucose, electrolytes and arterial blood gases were normal. He was given 5 mg diazepam intravenously which had little effect, and, if anything, the force of the spasms increased.

He remained in this state for about 100 minutes before he awoke, only to again lapse into unconsciousness for a further 5-minute period.

When he was questioned later, the patient admitted that there was a period during this time in which he was aware of people talking to him, but was totally unable to respond. He remained drowsy for a further few hours after regaining consciousness the second time. His subsequent hospital stay was then uneventful. Our patient was also unpremedicated and anxious. Saunders and Harris proposed that this state may be a precipitating factor for strange neurological events following propofol anaesthesia and that propofol should possibly be avoided in such patients.

We believe that this further case strengthens their argument.

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Hypercapnia and raised cerebrospinal fluid pressure

Subarachnoid anaesthesia is a technique ideally suited to lower abdominal and urological surgery in patients with reduced ventilatory reserve, especially those with CO₂

retention. An unusual finding in these circumstances is reported.

A 77-year-old man who had severe chronic obstructive

airways disease presented for transurethral resection of a bladder tumour. Blood gas analysis revealed a PCO_2 of 7.5 kPa and a PO_2 of 5.4 kPa. Subarachnoid anaesthesia was planned and the patient placed in the left lateral position. A 25-G spinal needle was inserted without difficulty into the subarachnoid space; clear fluid was seen in the hub. 0.5% hyperbaric bupivacaine 1.5 ml was injected via a 2-ml Plastipak syringe. It was noticed at the end of injection that fluid flowed back into the chamber of the syringe even if no pressure was applied to the plunger. Measurement via manometer tubing showed a pressure in excess of 4.0 kPa. The operation proceeded smoothly and no unusual symptoms were reported by the patient.

Westlake¹ investigated emphysematous patients with acute respiratory infections and found cerebrospinal fluid (CSF) pressures of up to 6.0 kPa. These patients exhibited signs and symptoms of raised intracranial pressure (headache, blurred vision, papilloedema). Newton and Bone²

described papilloedema and optic atrophy in two patients with chronic hypercapnia; the brain scan appearances were consistent with cerebral oedema.

The causes of raised CSF pressure in hypercapnia are mainly cerebral vasodilatation and cerebral oedema. It is uncommon, but the possibility of raised intracranial pressure should be considered when subarachnoid anaesthesia is performed in the hypercapnic patient.

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Whitacre and pencil-point spinal needles: some points to consider

The renewed popularity of spinal anaesthesia has led to the increasing use of fine gauge needles in an attempt to reduce the incidence of postspinal headache. Other factors are also involved, but both clinical and laboratory evidence indicate that the incidence and severity of this type of headache is proportional to the rate of leakage of cerebrospinal fluid through the hole in the dura mater. Most needle manufacturers now make needles as small as 26-gauge, but unfortunately in those groups particularly susceptible to postspinal headache, young and middle-aged adults, there are several reports of an incidence of 3-10% even with these fine needles, and some of these headaches may be severe enough to require an epidural blood patch.

These high susceptibility groups include obstetric patients, in whom postspinal headache is particularly distressing, and is largely the justification for the use of even finer needles. Most reports have been on 29- or even 30-gauge needles¹⁻³ and while the incidence of postspinal headache with these is very low or absent they present both practical and manufacturing difficulties. There is a loss of feel, a danger of bending or damage even when introduced through a larger needle or introducer and, unless aspirated, cerebrospinal fluid is slow to appear at the metal hub. In addition, no needle manufacturer has yet safely bonded such fine needle shafts to transparent plastic hubs, a considerable disadvantage when the hub represents such a high proportion of the needle's internal volume.

These difficulties have led to the renewal of interest in alternative needle tip design as an approach to the reduction of cerebrospinal fluid leak after dural puncture. As long ago as 1926 Greene⁴ advocated the rounding of the normal cutting point to separate rather than cut the dural fibres. In 1951 Haraldson⁵ (giving credit to Sjovall) and Hart and Whitacre⁶ suggested the use of pencil-point or conical-tipped spinal needles. These two designs were similar, except that in the former the needle orifice was on the cone-shaped tip, while in what came to be called the 'Whitacre' needle, it was on the shaft just proximal to the tip.

The original Whitacre needle, which was 20-gauge, was claimed to have reduced the incidence of spinal headache from 5-2% in an unselected group of patients and in 1960 Cappe⁷ reported an incidence of 0.63% in 318 obstetric patients using a 22-gauge version. Despite these reports, needles with cutting tips remained the most popular and some more recent reports have found a rather higher

incidence of postspinal headache with the larger pencil-point needles. The theoretical advantages of even smaller pencil-point needles (24 to 26 gauge) are that they might combine the low incidence of headache of very fine cutting needles (e.g. 29-gauge) with the ease of use and manufacture of larger needles, in particular making possible the provision of a transparent hub.

One of the first of the 'new generation' of pencil-point needles was the Sprotte 'atraumatic' needle which was recommended for a wide variety of regional anaesthetic uses.⁸ A claimed incidence of headache of 0.02% was reported using a mixture of 22-gauge and 24-gauge spinal needles for various types of surgery but excluding obstetrics, while Cesarini and his colleagues⁹ reported no postspinal headaches in 55 cases in which 24-gauge Sprotte needles were used for spinal anaesthesia for Caesarean section. Sprotte and his colleagues drew attention to the difference in design between their needle and the Whitacre needle. The former needle has a notch-like orifice with a diameter at least equal to the internal diameter of the needle, while the Whitacre needle had a small lateral hole with a diameter less than the internal diameter of the needle. Not only did this slow the appearance of cerebrospinal fluid at the hub, but it produced a markedly directional flow of local anaesthetic from the orifice and this was so pronounced that Graham and colleagues¹⁰ showed a significant difference in sensory levels between groups of patients in whom the orifice was directed cephalad or caudad for a Caesarean section, analgesia being inadequate for surgery in 80% of the latter group.

In the case of the Sprotte needle with its larger orifice, injected fluid is claimed not to be directed laterally but to continue in the line of the needle shaft, as is the case with cutting needles. We have made a preliminary investigation of this and other pencil-point needles by injection of dye-containing solutions through these into a fluid-filled glass spine. This indicated that while the modern needles inject the solution much more nearly in the line of the shaft than did the original Whitacre needle, there was some deviation of the injected fluid which might be of clinical significance when combined with other factors, such as the rate of injection and the angle of needle insertion. We intend to repeat these tests under more precise conditions.

None of the new needles appears to suffer seriously from the disadvantages of the original Whitacre needle, so it seems more important than simply a question of semantics

to differentiate between them. This author considers it correct to call them pencil-point or atraumatic, but preferably not Whitacre.

These are interesting points to consider.

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An adapter for a microcatheter

We were unable to reconnect a 26-gauge spinal catheter to its adapter (CoSpan Catheter; Kendall Health Products Company, Mansfield, MA) during an operation. General anaesthesia had then to be induced because injection of spinal anaesthetic was impossible.

Spinal anaesthesia with intermittent injections through a 26-G catheter was uneventful until the small extra pressure for injection caused disruption of the catheter from the adapter. It seems that the adapter-catheter connexion needs to be improved.

We found a solution to the problem later: the adapter from the Portex Epidural Minipack also fits a smaller diameter catheter, like the 26-G spinal microcatheter, and allows the injection of anaesthetic solution through it.

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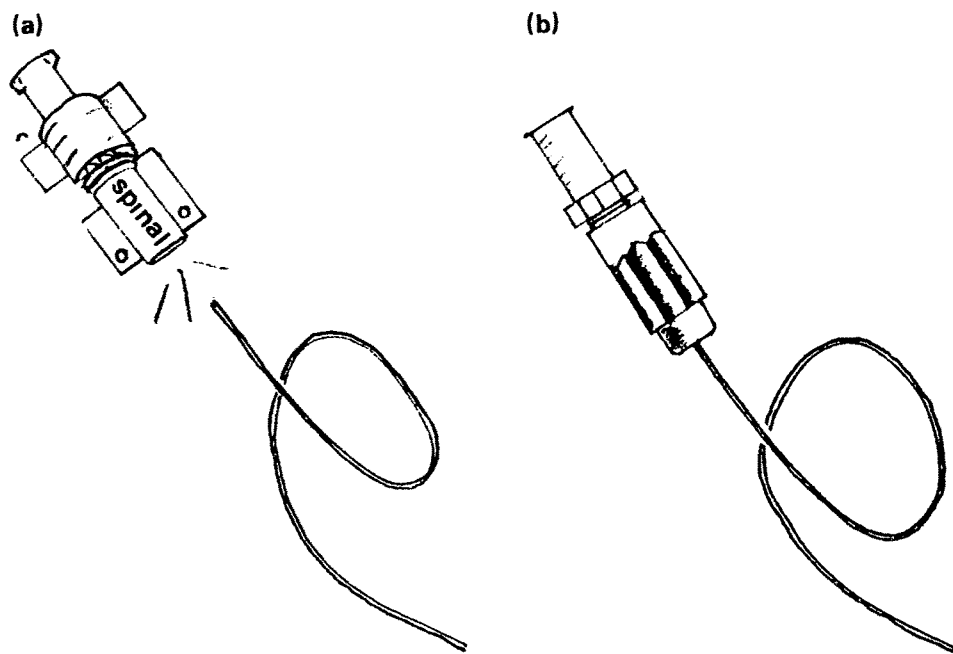


Fig. 1. (a) Kendall continuous spinal catheter adapter; (b) Portex epidural catheter adapter.

Too long plastic obturator of 18-G Portex epidural needle

The Portex epidural needle is well known for its quality. However, recently we found a needle which uneventfully 'passed' the quality examination of Portex personnel in which the plastic obturator of the 18-G epidural needle was

longer by 4 mm than the needle itself. This could allow a part of the plastic obturator to remain in a patient's body.

Examination before use of an epidural set can take some seconds but it is the quality test of the anaesthesiologist.

We should not rely on the manufacturer; this is not logical and is also illegal.

How many anaesthesiologists examine whether the epidural catheter really has three open holes?

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A reply

Portex epidural products are designed to comply with the relevant British Standards specification (BS 6196: 1989). This requires that the end of the stylet 'does not protrude from the needle point' (BS 6196: 1989).

Others report¹ that the design of the Portex product compares favourably with those of other manufacturers,

having a stylet which fills the lumen of the needle so as to prevent coring and is flush with, but does not protrude beyond, the needle point.

Unusual circumstances have caused the unit described to be defective. We are modifying our manufacturing processes in order to prevent recurrence.

The authors remind clinicians of their responsibility to check all equipment prior to use, irrespective of their confidence in its general quality. We endorse this view.

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Late postspinal headache treated with epidural morphine

We reported¹ six patients (28-70 years old) with postspinal headaches (PSH) in whom total relief of pain was obtained by epidural injection of morphine via an epidural catheter 24-36 hours from the dural puncture.

This case is the first of postspinal headache treated by epidural morphine 5 days after the inadvertent spinal.

A 57-year-old patient, who weighed 90 kg, was scheduled for lithotripsy. She had hypertension and was treated by nifedipine 10 mg three times per day. Her blood pressure on admission was 180/85 mmHg. Her haemoglobin was 13.8 g/100 ml, white blood cells 4800, and thrombocytes 110 000. No premedication was given. Epidural anaesthesia was planned with a Tuohy needle 18G (Portex) at the L₃₋₄ interspace. However, inadvertently, CSF was obtained and spinal anaesthesia with 10 mg bupivacaine 0.5% was substituted. The blood pressure decreased to 140/80 mmHg and remained so during the 45-minute procedure.

She received 3000 ml compound sodium lactate for 24 hours after the procedure. Several hours after the inadvertent spinal she complained of severe bifrontal and occipital headache with dizziness, nausea and vomiting. The headache was worse when she stood up or sat in bed. She refused treatment by epidural injection of blood, saline or morphine for the next 3 days. Epidural morphine was suggested because of a good previous experience with six patients.¹ She agreed to the treatment on the fifth day. An epidural catheter was introduced into the L₃₋₄ interspace, and 4 mg morphine diluted in 4 ml saline 0.9% were injected into the epidural space through the epidural catheter.

Her bifrontal headache disappeared completely 15 minutes after the injection; her occipital headache responded after 30-45 minutes. The patient sat in bed without dizziness, nausea or vomiting and was released from the hospital 18 hours afterwards. There was no headache for the next week.

There have been several reports on the successful use of epidural morphine after inadvertent spinal tap to prevent

postspinal headache,^{2,3} but this is the first report of the beneficial use of this modality after headaches had already occurred for 5 days.

Epidural morphine has some advantages over the usual methods of treatment because it causes analgesia and can prevent the PSH. It can be given shortly after the dural puncture, in contrast to epidural blood patch which often fails when given early.^{4,5} Morphine can be given through an epidural catheter which is left in place and can be given repeatedly without inconvenience to the patient.

We suggest that an epidural catheter be left in place after spinal anaesthesia, in case a PSH develops and for routine treatment after operation. We have used an 18-G epidural needle brazed to a 20-G spinal guide needle for combined spinal and epidural anaesthesia and for this purpose.

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25- or 26-G needles for spinal anaesthesia in young patients?

We would like to report the results of our retrospective study which was designed to answer this question by means of a questionnaire.

Three hundred and sixty-five out of 425 patients, aged 14 to 51 years, who had spinal anaesthesia performed for

various surgical procedures, answered our questionnaire about postoperative headache. Eighteen (4.9%) developed a well defined postspinal puncture headache (PSPH) (severe or intolerable, dependent on posture).

A 25-G (0.5 mm) spinal needle was used in 72 patients

for the anaesthesia, and in 258 a 26-G (0.45 mm) needle; no information on needle size could be obtained for 35 patients. Seven patients in the 25-G group (9.7%) and 11 (4.3%) in 26-G group had a PSPH. The two groups did not differ with respect to age or sex. Fisher's exact test gives $p = 0.14$ for the probability that the difference in rate of PSPH is due to chance.

There is one other study which compares these two needles with respect to PSPH.¹ However, no figures are given and no conclusive result is presented. Several studies which compare needles with greater differences in diameter have been performed,^{2,3} and show results in favour of the smaller needles with respect to PSPH.

The probability of 0.14 means that there is a risk of 14% that the difference is due to random chance. A prospectively performed study, designed to demonstrate a similar difference in PSPH rate would require 2×200 patients at the (usual and quite arbitrary) 0.05 significance level. A reduction in PSPH-rate of about 50% when using the 26-G needle instead of the 25-G is possible.

The 26-G needle is not more expensive than the 25-G needle and, in our personal experience, it is as easy to use.

We do not hesitate despite the high p-value, on the basis of our data, to recommend the use of the 26-G needle in preference to the 25-G needle.

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Spinal anaesthesia in obstetrics: headache and special needles

The efforts of Drs T.A. Thomas and H.A. Noble (*Anaesthesia* 1990; 45: 489) and their renewed examination of spinal anaesthesia in obstetric practice and their search for ways to reduce the incidence of postlumbar puncture headache are applauded. Like them I have vacillated between spinal and epidural in obstetrics.

Continuous caudal or lumbar epidural analgesia is commonly used for the relief of pain in labour but I have, for several years, changed more and more to spinal (subarachnoid) for Caesarean sections unless the patient prefers general anaesthesia or, for some reason (sometimes because she had an epidural before), refuses a spinal and insists on an epidural. A spinal is usually so easy, so fast, and so painless to administer, especially if the patient is sitting and flexed (so that the midline is easy to find). It works quickly, one does not have to wait 10-30 minutes to decide if one has a failure (might the failure rate even be lower than epidural?). The total dose of anaesthetic drug is about one-tenth of that used in caudal or lumbar epidural so, in theory, that should have less effect on the newborn and less chance of maternal convulsions.

The only good thing about epidural is the lower risk of headache (unless there is an unintentional dural puncture, in which case there is a very high chance of headache!).

Another major reason why some anaesthetists eschew spinal in obstetrics is that the rapid onset of extensive sympathetic blockade can quickly lead to hypotension, nausea, and retching. But this can easily be prevented by preloading the patient with a litre of intravenous fluid (warmed, to avoid shivering), prophylactic doses of intravenous ephedrine 10-20 mg at a time, and arrangement so that the patient's pelvis is tilted to the left.

It is important not to wait for hypotension to occur; if one does not prevent it, it will always happen with the high level of block needed to provide pain-free conditions (T_{2-4}). One should administer increments of intravenous ephedrine before hypotension happens! Intravenous metoclopramide 10 mg given before the spinal seems to make nausea and retching less likely, but there is little doubt that if one's patient is retching, the most likely cause is hypotension.

Four patients in a personal series of 120 Caesarean sections done over the last few years with spinal had a complete failure and general anaesthesia became necessary.

One other patient tolerated the first part of the operation but asked to be put to sleep after the baby was born. Thus, in 96% cases a spinal provided perfect, or almost perfect, anaesthesia.

Most of these patients had amethocaine (tetracaine, Pontocaine) 1% with dextrose (not available in the UK) but recently bupivacaine 0.5% plain, in a volume based on the height of the patient, between 3.0 and 4.25 ml, is used in the sitting position (the specific gravity of 0.5% bupivacaine is 1.007; CSF 1.003).

The addition of small amounts of morphine (0.25 mg) or fentanyl (10 µg) to the spinal anaesthetic seems to have improved operative anaesthesia; and, in the case of morphine, analgesia for 12 to 24 hours but 25% patients complain of itching.

The incidence of headache in my series is lower than Thomas and Noble who reported 22% in their unrestricted group and, 35% in their bed-rest group.¹ Three percent of my patients complained of headache and 1 (0.8%) required an epidural blood patch.

The needle was 25-gauge, of the old-fashion cutting bevel type (with the bevel horizontal, which I now realise might be wrong; it should have been vertical)² and no restriction was placed on the patients' activity.

This is my personal experience with a conventional needle; it will be very interesting to learn about a larger series of the 22-gauge Whitacre needle. If they have good results, they might try for even better ones, by using the 24-gauge version Sprotte needle which has recently become available (Pajunk GmbH, AM Holtzplatz 5-7, D-7716 Geisengen).

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A reply

Thank you for giving me the opportunity to comment on Dr Thomas' letter.

It is encouraging to receive the support of other practitioners and to hear of their experiences with subarachnoid anaesthesia for Caesarean section. My co-authors and I were interested to read of his use of warm intravenous fluids. We have encouraged for some time the use of a blood warmer in order to increase the temperature of the preload solution and believe it to be a useful refinement to the technique. The chilling factor of 1 litre of crystalloid at room temperature amounts to approximately 17 000 calories. Dr Thomas also makes useful practical points about the use of ephedrine and metoclopramide in the obstetric patient undergoing operative delivery.

We do, however, have very great reservations about his regimen of plain bupivacaine for these patients. Russell¹ has written extensively on the subject and indicates that plain bupivacaine solutions can produce an unpredictably high block in doses of 1.5-3 ml in pregnant patients. Our experience with the hyperbaric solution matches that of Russell. Indeed we find that we obtain a few high blocks with the hyperbaric solution in doses of 2.5 ml. We are therefore alarmed at the suggestion that 4.25 ml of the plain solution should be used, especially with the patient in the sitting position. The observation that the specific gravity of plain bupivacaine 0.5% is 1.007 is true at room

temperature. However, at 37°C, the specific gravity has fallen to 0.99, making the warm plain solution slightly hypobaric with respect to cerebrospinal fluid. One would expect this combination of large dose, hypobaricity and the sitting position to lead to a very high incidence of high blocks. There may be racial differences between the patients in the United States and the patients in the United Kingdom, but certainly on this side of the Atlantic we would be very worried about the use of such a combination.

The use of opioids in subarachnoid injections is a difficult and controversial area. Morphine, with its relatively low lipid solubility, seems to us to be an inappropriate drug for subarachnoid and epidural use. Fentanyl is a much more appropriate agent for this route of administration and, in the United Kingdom, diamorphine is another suitable agent with high lipid solubility which we have used successfully as an adjuvant to epidural bupivacaine. It is perhaps also worth pointing out here that the opioid itch responds well to small doses of naloxone, in many cases without any loss of analgesia.

Finally, we point out that the report of postspinal headache was in fact made by Dr E.A. Thornberry together with one of us (T.A.T.) and that the letter which reported the use of Whitacre needles was by us.

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H.A. NOBLE
T.A. THOMAS

Reference

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Omeprazole and ranitidine

Doctor Ewart *et al.* claim (*Anaesthesia* 1990; **45**: 527-30) from their comparison of omeprazole and ranitidine that 'omeprazole was more effective and consistent than ranitidine at maintaining gastric pH greater than 3.5'. This would, of course, be of considerable interest if proved

correct. Unfortunately, an equally valid hypothesis from the study is that 150 mg of ranitidine is an inadequate dose.

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D. SALMON

Anxiety before operation and serum potassium

The study by McClean and Watters (*Anaesthesia* 1990; **45**: 583-5) suggested that a small decrease in serum potassium concentration occurred in patients who were anxious during the 24 hours before surgery.

Potassium concentrations were measured in the afternoon before surgery, but the timing of surgery was not noted; presumably it occurred in both morning and afternoon. Timing is important in the interpretation of the results of potassium measurements, since there is a variability of 3.1% in serum concentrations with a lower mean value of 4.26 mmol/litre in the afternoon compared with 4.40 mmol/litre before 1300 hours.¹

Changes of 0.1 mmol/litre were deemed to be significant. This was based upon consideration of analytical imprecision alone. However, changes in serial results are due to pre-analytical sources of variation (including the sampling by different techniques such as conventional venepuncture or through a cannula) and inherent within-subject random variation as well as analytical imprecision. The average within-subject variation is 4.8% for potassium (as coeffi-

cient of variation) and is likely to be greater over short time periods, such as this study, because of serial correlation between values. Thus, for a change to be significant (at $p < 0.05$), it must be greater than 14% (approximately 0.5 mmol/litre). Biological variation is of much greater significance than analytical imprecision.

Knowledge of analytical imprecision and biological variation allows calculation of the probability that a decrease in serum potassium of 0.15 mmol/litre is significant; this probability is low, namely < 0.3 .²

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S.L. CROFTS
C.G. FRASER
I.G. SKIPSEY
R.E. WEBSTER

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2. FRASER CG, FOGARTY Y. Interpreting laboratory results. *British Medical Journal* 1989; **298**: 1659-60.

A reply

We investigated the relationship of changes in anxiety with changes in serum potassium concentration. It is known that adrenaline has an effect on serum potassium concentrations and the clinical relevance was investigated in this study.

Timing does have an effect on potassium concentration and we confirm that the samples taken immediately before surgery were obtained in the morning.

Many factors influence the potassium concentrations including inherent within-subject random variation and analytical imprecision (although the effect of the different

methods of obtaining a sample of blood are not proven).¹ The conclusions of this study are that no clinically significant change in potassium concentration occur in normal patients in the peri-operative phase of anaesthesia as a result of change in anxiety, and the writer's points on analytical and within-patient variation are well taken but do not alter this conclusion.

Craigavon Hospital,
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G. McCLEANE

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1. FELL D, DERBYSHIRE DR, MAILE CJD, LARSSON I, ELLIS R, ACHOLA KJ, SMITH G. Measurement of plasma catecholamine concentrations. An assessment of anxiety. *British Journal of Anaesthesia* 1985; **57**: 770.

Goldenhar's syndrome

This is a response to Dr Madan's reply to my letter (*Anaesthesia* 1990; **45**: 593).

My patient was 2 months old and weighed 4.5 kg (not 9.5 kg); this was my error for which I apologise. There was no difficulty in tracheal intubation on the second and third attempts.

We did not have a capnograph or pulse oximeter at that time, so the only alternative was to confirm tracheal intubation by seeing the tube pass through the cords, auscultation of the chest, observing the movements of reservoir bag and the absence of cyanosis.

Bradycardia persisted as long as the tracheal tube was in place and did not respond to three doses of atropine 0.01 mg/kg intravenously. Ketamine was preferred for its sympathomimetic and analgesic effects.

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F.M. KHAN

Inadequate reversal

The use by Dr Kumar of the laryngeal mask for inadequate reversal was interesting (*Anaesthesia* 1990; **45**: 792). However, could the problem not have been avoided by the use of a nerve stimulator? The correspondent mentions four monitors used during the operation but not, as it turned out, the vital (and cheapest) one.

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J. CADDY

Mouthwash sweetens ketamine anaesthesia

A 2-year-old boy with acute lymphoblastic leukaemia received ketamine anaesthesia daily for one week, and twice daily for the second week for radiotherapy. He was unpremedicated, and had two indwelling tunnelled central venous lines, through which the anaesthetic drugs were given. We noted, at the beginning of the second week, that although he arrived in the treatment area calm and cheerful, a few seconds after the injection of ketamine was started he would wriggle, grimace and protrude his tongue as if he had an unpleasant taste. Recovery was invariably peaceful, so fear of dreams seemed unlikely, and use of a

different antisialogogue made no difference. Abnormalities of taste are not normally associated with ketamine. The boy's father, who always accompanied him, confirmed that the little boy did have a nasty taste on induction, and gave him a mouthwash of benzydamine (Diffiam, Riker) one hour before treatment. Induction of anaesthesia was subsequently far smoother, with giggles instead of grimaces.

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A. DAVIES

A drawover breathing system for weaning

We exhausted our supply of disposable condenser humidifiers which we had found particularly useful for humidification of intubated patients during weaning from mechanical ventilation. We have no access to a blower humidifier

system to supply a T-piece, so we adapted one of our heated humidifiers to a drawover system comprising a non-rebreathing valve, ventilator tubing, humidifier, oxygen T-piece and reservoir, to allow humidification for an intu-

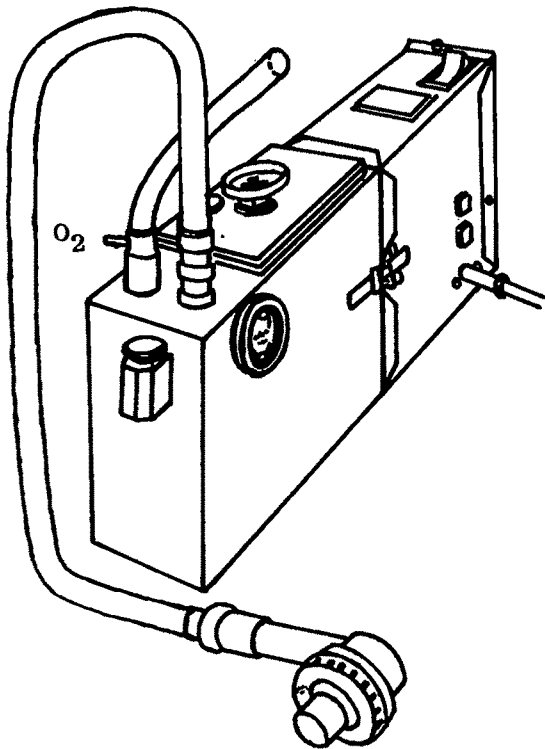


Fig. 1.

bated, spontaneously breathing patient. We use a Cape autoclavable humidifier, which works well in practice, although we do not use it for small children due to the added resistance of the non-rebreathing valve.

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Zambia*

M.J. BEM
I.H. WILSON

Deliberate oesophageal fiberoptic intubation

We have used this method on two occasions in patients whose airway was already secured by a tracheal tube. A nasogastric tube was required and the familiar techniques failed. A small (6.5 mm) tracheal tube was passed over the fiberoptic laryngoscope through the nose. Tracheal intubation was reconfirmed and then the fibroscope was passed into the oesophagus. The small tracheal tube was passed into the oesophagus and the laryngoscope withdrawn. A nasogastric tube was then passed into the oesophagus and thence into the stomach.

This technique seemed quite easy on these two occasions and others might find the method useful.

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D.P. BREEN
S.P. GERRISH

A novel method of pre-oxygenation

A patient was to have a period of pre-oxygenation. A previous experience had left her in fear of an anaesthetic mask and she refused to cooperate. The procedure was modified by placing a Pall heat and moisture exchanger on the breathing system (in this case a Bain system) with the patient using the open-end as a mouthpiece.

The nose was gently occluded. This enabled effective pre-oxygenation and the patient cooperated throughout.

This technique is an alternative to the traditional face-mask for an anxious patient.

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S.P.S. CHEEMA
C.S. EVANS

Book reviews

Memory and awareness in anaesthesia	1104
Edited by B. BONKE, W. FITCH AND K. MILLAR	
Textbook of anaesthesia, 2nd edn	1104
Edited by A.R. AITKENHEAD AND G. SMITH	
Ostlere and Bryce-Smith's anaesthetics for medical students	1105
T.B. BOULTON AND C.E. BLOGG	
Manuel d'anesthésie pédiatrique	1105
C. SAINT-MAURICE, I. MURAT AND C. ECOFFEY	

Resuscitation handbook	1106
P.J.F. BASKETT	
Anaesthesia, 3rd edn	1106
Edited by R.D. MILLER	
What price quality. The NHS in review	1106
G. McLAUCHLAN	

Memory and awareness in anaesthesia

Edited by B. BONKE, W. FITCH AND K. MILLAR. Pp. 394. Swets and Zeitlinger, 1990. Dfl. 75.00.

This book comprises the proceedings of a joint Symposium of anaesthetists and psychologists held in Glasgow in 1989. The Symposium itself had been inspired by an article, published in a leading medical journal, which was heavily critical of the methodology, analysis and interpretation of many studies which claimed to demonstrate evidence of subconscious memory for events which occurred during anaesthesia. The book contains a number of contributions which validate that criticism.

The book is divided into four sections, dealing with psychological testing in relation to memory and awareness, psychological consequences of anaesthesia and surgery, effects of specific drugs, and monitoring of 'depth' of anaesthesia. Each section contains a mixture of review articles and 'original' scientific papers. Many of the latter have been published elsewhere. At least one has been printed in an almost identical form in a scientific journal since publication of the book; this highlights a dilemma which editors of journals and editors of Symposia Proceedings should address.

In general, the review articles are useful, and analyse existing knowledge critically and constructively. As is to be expected, the quality of the scientific contributions varies widely. The terms 'memory' and 'awareness' have clearly been used liberally by some contributors. One chapter consists of 'a report of 1000 anaesthetics' (in 557 patients) in which patients were asked at a pre-operative visit to relate their recollection of events associated with previous operations. Virtually all of the recollections related to postoperative sequelae. Two patients recalled intra-operative events, but these are dismissed by the authors as being 'a result of insufficient anaesthesia (rather than) real cases of awareness'. Scientific excellence appears not to have been a prerequisite for publication. One paper concludes that the anaesthetic technique which the authors investigated resulted in 'most importantly, no signs of awareness or recall'; in total, 11 patients were studied.

The main emphasis of the Symposium was awareness without recall, and the majority of contributions relate to the various methods by which 'unconscious perception' can be demonstrated (or not). Anaesthetists who have a special interest in awareness *with* recall after anaesthesia will find little in the book to advance their knowledge of the incidence, causes or prevention of this complication. However, for those interested specifically in memory

processes during anaesthesia, and in experimental design in this field, the papers based on the main Conference Addresses, and some of the other review articles, provide useful summaries of existing knowledge and a host of key references; the papers by Kihlstrom and Bonke are particularly worthwhile. There is also a very interesting contribution from Levinson, a pioneer of research into unconscious perception, which provides some fascinating background facts relating to his classic, but unrepeatable, study of hypnotised patients. The remainder of the book may provide some entertainment, but little new information.

A.R. AITKENHEAD

Textbook of anaesthesia, 2nd edn

Edited by A.R. AITKENHEAD AND G. SMITH. Pp. xii + 773. Churchill Livingstone, 1990. £32.50.

This is a second edition, 5 years after the first, and includes a major revision. A number of the existing chapters have been rewritten by new authors, including the editors, with an excellent chapter in particular on haematology. Additional chapters cover basic physics, postoperative care and fluid balance whose mysteries are very clearly explained.

The book is aimed at the new recruit to the specialty in the first 2 years of their training and those who wish to take Part I FCAnaes examination. The editors have attempted, therefore, the difficult task of covering all subjects at an early level. The first half of the book is devoted to the scientific fundamentals; it is a good principle to cover the physiological and pharmacological groundwork. Some topics are taken to a higher level than is required for the Part I FCAnaes examination but probably still insufficient for the Part II. Some chapters could be improved by the use of the opportunity to spell out certain basic facts since, very often, the early trainee has forgotten fundamental chemical knowledge which was last taught some years earlier at school.

The space given to the historical volatile agents is still more than is required to illustrate the basic physical principles which are important.

The clinical part of the book attempts to cover the whole range of topics including basic technique, emergency anaesthesia and virtually all the specialties. The chapters dealing with fundamental techniques and emergency

procedures explain very clearly what is to be done in emergency situations and can claim some of the credit for the sound performance of many candidates in the examination. It is difficult to write a chapter on the basic conduct of anaesthesia. This chapter is clear but the author has missed the opportunity to discuss the positioning of the patient on the operating table and the relevant problems and complications of each position. There are a series of clinical chapters about anaesthesia for dental, ENT, and gynaecological surgery. Ideally, at this level the authors should explain common basic problems rather than attempt to cover the whole field. Some have been successful, but there are exceptions, for example, where there is only a single paragraph about anaesthesia for fractured neck of femur in the elderly! Chapters on anaesthesia for neurosurgery, cardiac, thoracic and neonatal surgery are irrelevant at this level. Anaesthesia for neurosurgery is a clear, well-written chapter but it is necessarily brief. This topic should be limited to the basic physiological and pharmacological information, together with the early management of patients with head injuries and anaesthesia for those who require other surgery. Authors could indicate where they have limited their discussion and direct the interested reader elsewhere.

The appendix at the end of the book is extremely helpful. Useful additions would include the conversion between cm H₂O and mmHg in the SI Unit appendix and the formulae for the calculation of length and diameter of a tube. Each chapter has a significant number of references for further reading but this list could be improved by an indication of the relevant reference within the text.

This textbook can be recommended strongly for all trainees in the initial part of their training. It is, quite deservedly, adopted by a number of departments around the world as the recommended text for their new recruits. However, it should not be regarded as a syllabus for the Part I FCAnaes examination.

F.J.M. WALTERS

Ostlere and Bryce-Smith's anaesthetics for medical students

T.B. BOULTON AND C.E. BLOGG. Pp. x+271. Churchill Livingstone, 1989. £6.95.

Drs Boulton and Blogg are credited as the authors of the 10th edition of this book, but the influence of Drs Ostlere and Bryce-Smith remains evident. The foreword draws attention to the change in attitude towards training and responsibility in anaesthesia, remarking that one of the 'junior' authors gave his first solo anaesthetic for an emergency obstetric procedure as a medical student in 1948. The authors note that the pattern of training of medical students has changed since 1948, when newly qualified doctors were expected to adopt immediate responsibility for many aspects of medical care, including the administration of anaesthesia. Now, undergraduate training is designed to provide a broad preview of medicine, and specialisation takes place after qualification. Unfortunately, this change in emphasis appears to have been overlooked by the authors in preparing the text, which, by their own criteria, is more appropriate to the expectations of the 1948 student.

The book starts with a brief history of anaesthesia (further details are provided in an appendix), and introduces the reader to anaesthetic 'jargon'. A chapter is devoted to the concepts of the different techniques of anaesthesia, and another to potentially dangerous events during anaesthesia, and their prevention. So far, so good.

However, the remainder of the book describes apparatus and techniques at a level far beyond that required by medical students. While an insight into the work of the anaesthetist is appropriate, the unnecessary detail provided in these chapters can serve only to confuse the student. In several places, the text is written in the form of instructions, appropriate only if the reader is expected to undertake the procedure himself. For example, in the section on paediatric anaesthesia, the reader is directed: 'Use thiopentone (3–5 mg/kg) or methohexitone (1.5 mg/kg)'. There are two pages of instructions on how to conduct rapid sequence induction. With regard to cardiac arrest during anaesthesia, the student is urged: 'Inform the surgeon and stop the operation. Discontinue the anaesthetic.' It is clear that many sections have been written as practical guides for practising, unsupervised trainees, and not with a view to assisting the modern medical student, who is primarily an observer, or at most a strictly supervised practitioner in the operating theatre, to understand current concepts of balanced anaesthesia. While teaching of practical skills is of importance, it is surely irrelevant, and potentially dangerous, to provide intricate and detailed instruction in the administration of anaesthesia.

There are in addition, frequent references to drugs and techniques which are rarely used in modern anaesthetic practice. There is an irritating use of colloquialisms, such as 'depolarisers'. The equally irksome anecdotal footnotes, many of which do little to enhance the image of anaesthetists in the eyes of potential recruits, have been retained from previous editions.

Many important and relevant issues are discussed, including pre-operative assessment and investigations, the effects of intercurrent disease on anaesthesia, and cardio-pulmonary resuscitation. Unfortunately, other pertinent subjects, such as postoperative pain relief and fluid balance, are ignored, and there is no mention of the role of the anaesthetist in the intensive care unit or chronic pain clinic.

A discussion document published by the College of Anaesthetists ('Academic Departments of Anaesthesia in Undergraduate Education: an Undervalued Resource') outlines the current philosophy of undergraduate teaching in Anaesthesia. It is to be hoped that Drs Boulton and Blogg are allowed to adapt the book to reflect modern teaching when they prepare the 11th edition.

A.R. AITKENHEAD

Manuel d'anesthésie pédiatrique

C. SAINT-MAURICE, I. MURAT AND C. ECOFFEY. Pp. 437+index. Pradel Paris, 1990.

This new French textbook on paediatric anaesthesia (as far as I am aware the first of its kind) marks the pinnacle of the distinguished career of Claude St Maurice.

The scope of this textbook is wide, with discussions about neonatal surgery and cardiac and liver transplant; however the basic differences and challenges of paediatric anaesthesia could have received more emphasis.

The book is divided into four parts; pre-operative preparation, pharmacology, pre-operative and post-operative care. There is no preface, and thus no definition of the intended readership. The recent Anglo-American literature in paediatric anaesthesia is well reviewed and should prove invaluable for French readers.

The book describes French methods which may not be the same as used in other countries. Early surgical intervention in congenital diaphragmatic hernia has lost popularity

in many other centres. Little emphasis is placed on the psychological development of children and hence their appropriate preparation for surgery.

Claude St Maurice, Isabelle Murat and Claude Ecoffey should be congratulated on this landmark French paediatric anaesthetic textbook, which can only raise interest in this field in France. However, the English-speaking anaesthetist has access to a larger range of books, not only on general paediatric anaesthesia but on specific areas such as cardiac and regional paediatric anaesthesia. This book should prove popular in France and French-speaking countries but may have limited appeal elsewhere.

A.E.E. MEURSING

Brief reviews by the Editor

Resuscitation handbook

P.J.F. BASKETT. Pp. 113 + index. Gower Medical, 1990. £9.95.

A handbook is intended to inform its reader what, how, and when to perform a task. This one does this for resuscitation by doctors and medical students by means of text, coloured diagrams and protocols. Plagiarism by teachers or instructors is encouraged by the provision at extra charge of 145 colour slides. The book is certainly worth its price, but the latter seem expensive at £1.03 p *each*.

Anesthesia, 3rd edn

Edited by R.D. MILLER. Pp. 2420 + Appendix and index. New York: Churchill Livingstone, 1990. £120.

It might seem churlish if we were not to note the advent of *Anesthesia* in its third edition. It differs so markedly from the second edition (two volumes instead of three) and in its genesis that bookworms should take note. There are now five additional consulting editors who must have been

amongst those whom the Editor (Dr R.D. Miller) consulted when the revision of this large book was contemplated. Indeed he states in the preface that no fewer than eight leaders in education in anesthesiology plus several members of the University of California reviewed the previous edition 'in depth'. These individuals were asked to comment on specific chapters from a position of considerable knowledge and to answer some very pertinent questions. The result is that 21 authors were dropped and 49 new ones were recruited; the resultant 80 chapters can legitimately be assumed to be the result of a peer review process second to none. No one individual could undertake to read every chapter of the published book and this individual has not attempted so to do. If I were faced with the prospect of purchase of one title to use for reference and study I think I would choose this one over any other similar compendium of information about anaesthesia.

What price quality? The NHS in review

G. McLachlan. Pp. 267 plus notes and two appendices. £15.00. The Rock Carling Fellowship, 1990. The Nuffield Provincial Hospitals Trust, London.

It has been my pleasure to review three or four monographs in this series during the past 8 years. They are not widely advertised and are easily overlooked. Gordon McLachlan served the Association of Anaesthetists of Great Britain and Ireland extremely well in his advocacy of the Trust's support for our first effort about the quality of anaesthetic services in the United Kingdom. Many anaesthetists are involved nowadays in management and this up-to-date monograph, written by an accountant of considerable experience of the National Health Service, should not be ignored. Even if you do not read all the book the chapters entitled *Research and health care* and *Perspective and evolution* towards the end deserve widespread attention.

J.N. LUNN

Anaesthetic literature

This section of *Anaesthetic literature* contains references from *Current Contents—Life Sciences* for July 1990. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, 145 Harley Street, London W1N 2DE. Dr Kaufman is prepared to provide on request, and at a modest charge, an additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, cpm 86 and on Dbase III, MsDos and are available on disc, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please. Readers should note, however, that this is the last occasion on which *Anaesthetic literature* will appear.

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Origination by BPCC Whitefriars Ltd, Tunbridge Wells, and printed and bound in Great Britain by
BPCC Wheatons Ltd, Exeter

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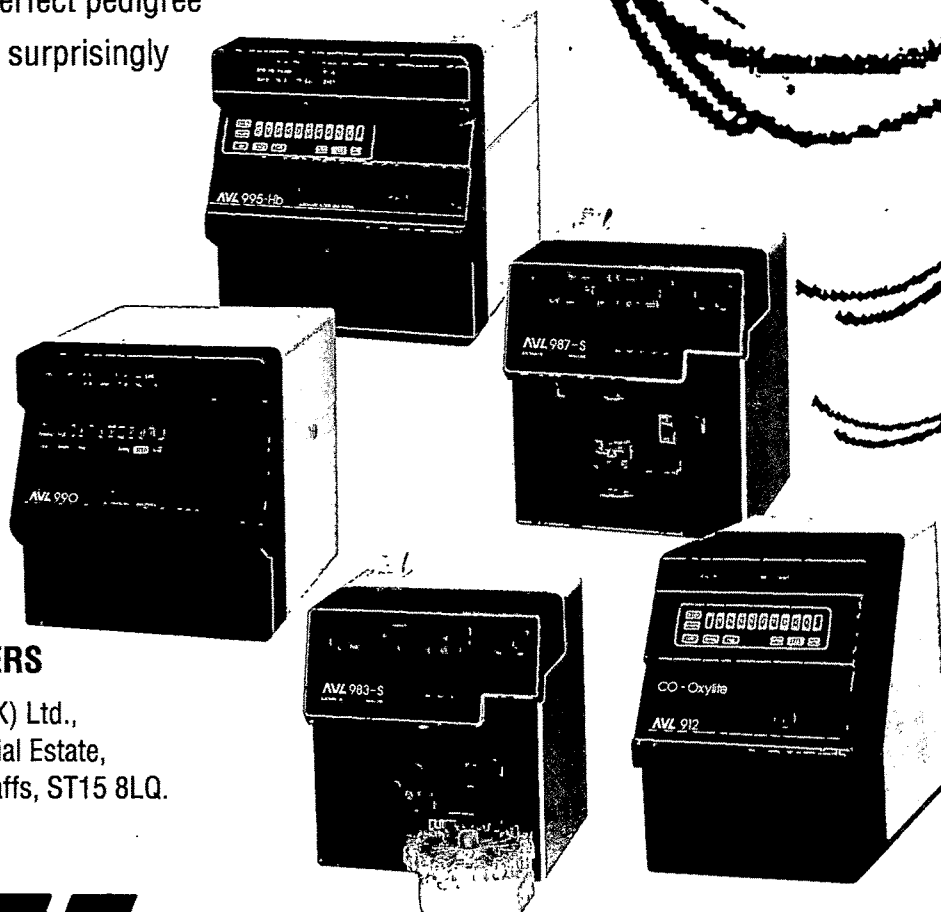
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